DISSERTATION ON

COMPARISON OF PROPOFOL AND ETOMIDATE INDUCTION ON HEMODYNAMIC RESPONSE TO ENDOTRACHEAL INTUBATION

Dissertation submitted in partial fulfilment of the regulations for the award of the degree of

M.D ANAESTHESIOLOGY BRANCH-X

OF

TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY
CHENNAI, TAMILNADU

ESIC- MEDICAL COLLEGE & POSTGRADUATE INSTITUTE OF MEDICAL SCIENCE AND RESEARCH,
KK NAGAR, CHENNAI-78

APRIL- 2019

ENDORSEMENT BY THE DEAN / THE HEAD OF THE INSTITUTION

This is to certify that this dissertation titled **"COMPARISON OF PROPOFOL AND ETOMIDATE INDUCTION ON HEMODYNAMIC RESPONSE TO ENDOTRACHEAL INTUBATION"** submitted by **Dr.JAMUNA S.** appearing for M.D Degree Branch–X ANAESTHESIOLOGY examination in April 2019, is a bonafide record of work done by her under my direct guidance and supervision in partial fulfilment of the regulations of Tamil Nadu Dr.M.G.R Medical University, Chennai. I forward this to the Tamil Nadu Dr. M.G.R Medical University, Chennai Tamil Nadu, India.

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This is to certify that dissertation entitled "**COMPARISON OF PROPOFOL AND ETOMIDATE INDUCTION ON HEMODYNAMIC RESPONSE TO ENDOTRACHEAL INTUBATION"** is a bonafide research work done by **Dr. JAMUNA S.,** in partial fulfillment of the requirement for the degree of M.D. in Anaesthesiology.

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This is to certify that the dissertation named **"COMPARISON OF PROPOFOL AND ETOMIDATE INDUCTION ON HEMODYNAMIC RESPONSE TO ENDOTRACHEAL INTUBATION"** is a bonafide work performed by **Dr. JAMUNA S,** postgraduate student, Department of Anaesthesiology, ESIC Medical college & PGIMSR, Chennai – 78, under my guidance and supervision in partial fulfilment of regulations of The Tamil Nadu Dr. M.G.R Medical University for the award of M.D. Degree during the academic year 2016 – 2019.

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DECLARATION

I solemnly declare that the dissertation titled **"COMPARISON OF PROPOFOL AND ETOMIDATE INDUCTION ON HEMODYNAMIC RESPONSE TO ENDOTRACHEAL INTUBATION"** has been conducted by me at ESIC Medical College & PGIMSR, Chennai, under the guidance and supervision of **Dr. Shirishkumar Chavan M.D.,** and **Dr. R.Uma M.D.,** Department of Anaesthesiology, ESIC Medical College & PGIMSR, Chennai. This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the University regulations for the award of the degree of M.D. Branch X (Anaesthesiology).

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ACKNOWLEDGEMENT

This study would not have the spirit it has without the invaluable academic, educational, psychological and human support and belief in me as a student and post graduate, provided by the following scholars.

I would like to convey my gratitude to our Dean **Dr. SOWMYA SAMPATH M.D (Paed), DNB (Paed).,** for providing me unflinching encouragement and support.

I am grateful to my Professor **Dr. SHIRISHKUMAR CHAVAN, MD.,** Head of Department of Anesthesiology and my guide for his supervision, advice and guidance from the very early stages of this study.

I am highly indebted to **Dr. R. UMA, M.D,** Associate Professor, my Co-guide and mentor, who had been instrumental in the completion of this study and for her supervision, advice and giving me moral support throughout the study.

I would like to thank **Dr. RADHIKA, M.D., Dr. ELANGO GANESAN, M.D.,** Associate professor and **Dr**. **ASHWINI, M.D**. Assistant professor who had been instrumental in completion of this study and for their supervision, advice and giving me moral support through the study.

I would also like to thank, **Dr. SHANMUGASUNDARAM**, HOD of The Department of General Surgery who had permitted to do the study on his patients.

I am grateful in every possible way to Assistant Professors, Specialists, Medical Officers, Senior Residents and the Postgraduates of the Department of Anesthesiology for helping me to conduct the study in the theatre. Many thanks in particular to the statistician **Dr. ARUNA PATIL, Ph.D.,** for her guidance regarding the sample size and data analysis.

I am thankful to the Institutional Ethical Committee for their guidance and approval for the study.

I also thank the theatre personnel for their help. And lastly but not the least, I would like to thank all the patients for their participation and cooperation and their trust in me.

I would like to thank my beloved husband and family for the support.

Dr. JAMUNA

CERTIFICATE OF APPROVAL

To

Dr. Jamuna S,

PG in Department of Anesthesiology,
ES IC Medical College & PGIMSR,
KK Nagar, Chennai-78.

Dear Dr. Jamuna S,

The Institutional Ethics Committee of ESIC Medical College &PGIMSR reviewed and discussed your application for approval of the proposal entitled "Comparison of Propofol and Etomidate Induction On Hemodynamic Response to Endotracheal Intubation", No.03-17/04/2017.

The following members of the Ethics Committee were present in the meeting held on 17/04/2017.conducted at ES IC Medical College &PGIMSR, KK Nagar,Chennai-78.

S.No.	ETHICS COMMITTEE MEMBERS
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12.	Dr. Napinai, Clinical Psychologist, EC Member
14.	Shri. K M Venugopal, Advocate, EC Member

The proposal is approved to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and significant adverse effects occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

A.V. SRINIVASA
HAIRPERSON

[DR. A.V. SRINIVASAN]
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This is to certify that this dissertation work titled " **COMPARISON OF PROPOFOL AND ETOMIDATE INDUCTION ON HEMODYNAMIC RESPONSE TO ENDOTRACHEAL INTUBATION"** of the candidate **Dr. Jamuna S.** with **Reg.No.201620501** for the award of Master Degree in the branch of Anaesthesiology. I personally verified the urkund.com website for the purpose of Plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and results shows 12% of Plagiarism in the dissertation.

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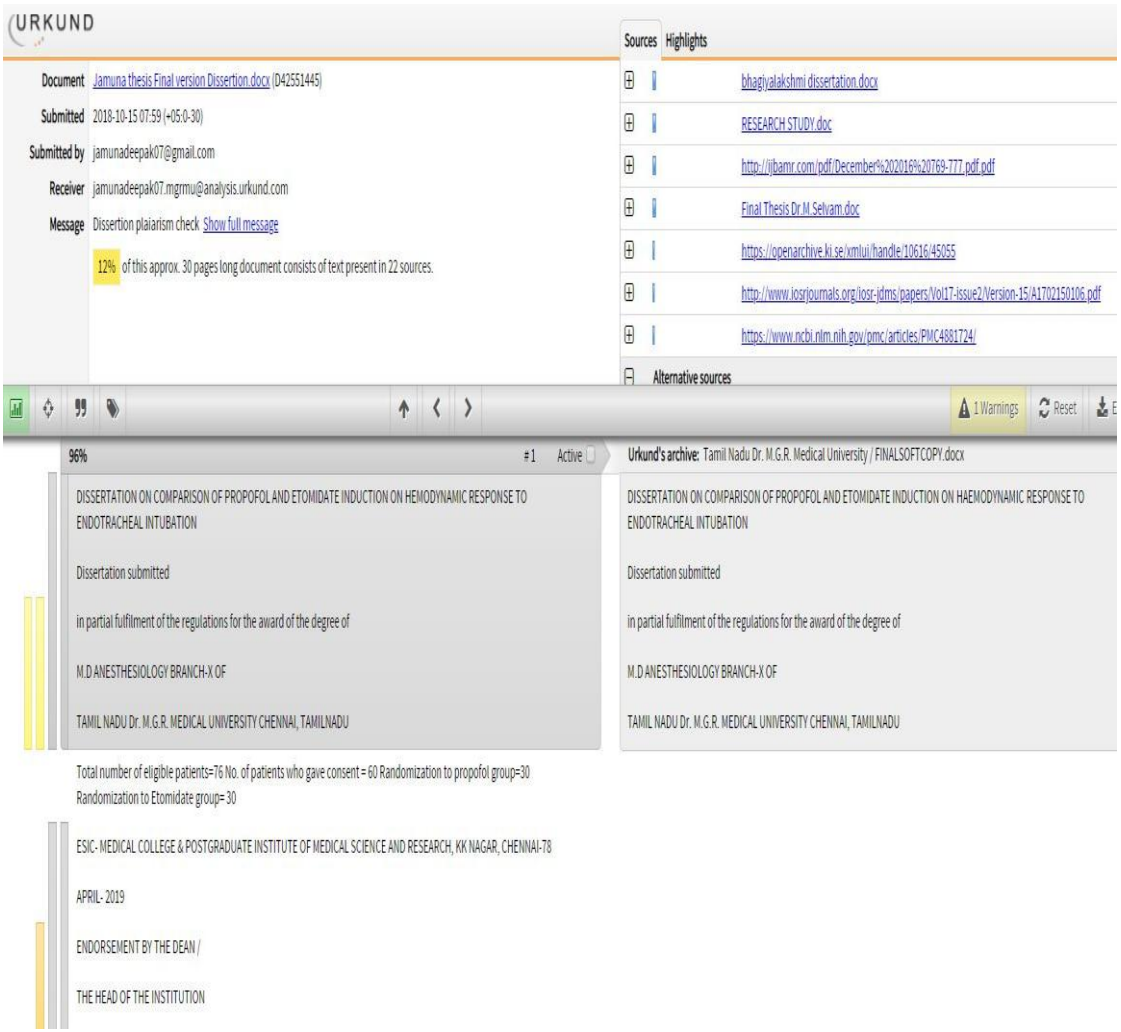
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TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY CHENNAI, TAMILNADU
Total number of eligible patients=76 No. of patients who gave consent = 60 Randomization to propofol group=30 Randomization to Etomidate group= 30
ESIC- MEDICAL COLLEGE & POSTGRADUATE INSTITUTE OF MEDICAL SCIENCE AND RESEARCH, KK NAGAR, CHENNAI-78
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LIST OF ABBREVIATIONS

Mg	→	Milligram
Mcg	→	Micrograms
ASA	→	American Society of Anesthesiologists
Cm	→	Centimeter
TC	→	Total Count
DC	→	Differential Count
Dl	→	Decilitre
ECG	→	Electrocardiogram
Gm	→	Gram
Hrs	→	Hours
NIBP	→	Non-invasive Blood Pressure
IM	→	Intramuscular
IV	→	Intravenous
Kg	→	Kilogram
Min	→	Minutes
Ml	→	Milliliter
mm of Hg	→	Millimeter of mercury
S	→	Significant
SD	→	Standard Deviation
SE	→	Standard Error
SBP	→	Systolic Blood Pressure
Mcg	→	Micrograms
NMDA	→	N-methyl – D- aspartate
GABA	→	Gamma-aminobutyric acid
CNS	→	Central Nervous System
Postop	→	Postoperatively
MAP	→	Mean Arterial Pressure
IVF	→	Intravenous Fluid
HR	→	Heart Rate
DBP	→	Diastolic Blood Pressure

CONTENTS

Introduction

INTRODUCTION

The introduction of general anesthetics into clinical practice date back to 150 years. It stands as one of the pioneering innovations of medicine, that lead to the development of modern surgery and spawned the speciality of anaesthesiology. General anaesthesia can broadly be defined as a drug-induced reversible depression of the central nervous system resulting in the loss of perception to all external stimuli. It is usually defined as a triad of amnesia, analgesia, and muscle relaxation.

Since introduction of general anaesthesia, no ideal induction agent has been discovered which provides stable hemodynamic conditions during endotracheal intubation.

Airway management and patient safety is the most important aspect of patient management in general anaesthesia. Safest and gold standard method of protecting airway, delivering anaesthetic gases and protection against aspiration is securing airway with endotracheal tube [1, 2].

An ideal induction agent should have hemodynamic stability and minimal intubation stress response, rapid clearance. The laryngoscopy

and intubation causes stress response leading to changes in hemodynamic parameters which can be detrimental to patients who are at cardiac risk[3].

These hemodynamic responses can affect myocardial perfusion in a negative way by increasing the myocardial oxygen demand and cardiac work load which can lead to ischemia.

During intubation, stimulation of laryngeal and tracheal tissues causes catecholamine discharge which can cause an increase in sympathetico adrenergic activity causing an increase in heart rate and systemic arterial pressure. Uses of general anaesthetic agent with intravenous induction can often cause hypotension by many mechanisms. Most important are suppressive effects of these agents on myocardial contractility, baroreceptor activity, sympathetic activity and central nervous activity.

Propofol is most commonly used agent for induction in general anaesthesia. It is a short acting IV anesthetic agent but it causes hemodynamic instability by causing profound hypotension. It also causes pain on injection. Allergic reactions are also being documented.

Etomidate is recently added drug to induction agent and being used in common practice in recent days due to its cardio stable nature.

It also has side effects like nausea, vomiting, increase in epileptogenic activity in patient with seizures, myoclonic activity. Rare but most important side effect of etomidate is decrease in serum cortisol by inhibition of 11 – beta–hydroxylase enzymes[(26)] even after single dose for up to 24 hours but this decrease was found to be within physiological range.

The primary objective of this study was to compare the efficacy of two different induction agents (Inj. Propofol and Inj. Etomidate) in maintaining hemodynamic stability during induction and following endotracheal intubation in elective surgery.

Aims
and
Objectives

AIMS AND OBJECTIVES

AIM OF STUDY

To compare the effect of intravenous induction agents propofol and etomidate in maintaining hemodynamic stability during and after endotracheal intubation.

OBJECTIVES

1) To assess the effect of the induction agent on the variation in heart rate during laryngoscopy and intubation.

2) To assess the variation of blood pressure during laryngoscopy and intubation.

Review of Literature

REVIEW OF LITERATURE

Ye L et al[4] [2017] , did a study of Comparison between Etomidate and Propofol Anaesthesia in Patients Undergoing Gastrointestinal Endoscopy: A Systematic Review and Meta-Analysis. 1115 patients were included in this meta-analysis based on six randomized controlled trials. There were no significant differences identified regarding duration of anaesthesia between etomidate and propofol group. Mean arterial pressure, recovery time, heart pulse, SPO2 at intubation, patient satisfaction, hypotension, changes of heart rate and nausea-vomiting were also comparable in both the groups. In Comparison with propofol, etomidate showed reduced apnea or hypoxemia and injection pain but with an increased myoclonus.

Baradari AG et al[5] [2017] did a randomized clinical trial comparing hemodynamic response to ketamine-propofol combination versus etomidate during anaesthesia induction in patients with left ventricular dysfunction undergoing coronary artery bypass graft surgery. In his study a total of 84 patients with ischemic left ventricular dysfunction (EF<40%) where randomly divided into 2 groups. Patients in group A received etomidate 0.2 mg/kg and placebo, whereas group B received ketamine 1mg/kg and propofol 1.5 mg/kg. Hemodynamic

variables systolic blood pressure, diastolic blood pressure, mean arterial pressure and heart rate were measured during and after laryngoscopy and before intubation at post intubation at 1, 2 and 3mins. The decrease in all hemodynamic parameters were greater in ketofol group (group B) than in etomidate group (group A) ($p<0.005$).They found that etomidate provides more hemodynamic stability when compared to ketofol in patients undergoing CABG surgery

Meena K, et al[(6)][2016] did a comparative study of effects of propofol, etomidate and propofol plus etomidate induction on hemodynamic response to endotracheal intubation: A randomized controlled trial. 90 patients in the age group of 15-60 years of either sex and ASA physical status I and II who were scheduled for elective surgery. Group 1- inj. propofol (2.5mg/kg), Group 2-inj.etomidate (0.3mg/kg), Group 3 - inj. propofol (1mg/kg) plus etomidate (0.2mg/kg). Systolic blood pressure, diastolic blood pressure, mean arterial pressure and heart rate were measured at variable intervals following intubation. In this study they founded that etomidate plus propofol has better hemodynamic stability than etomidate alone at 1 min , though etomidate was equally stable at other points.

Aggarwal et al[7] [2016] did a comparative study between propofol and etomidate under general anaesthesia. He studied 100 ASA I and II patients in the age group of 18 to 60 who were scheduled for elective procedure under general anaesthesia. 100 patients were divided randomly in 2 groups receiving propofol (2mg/kg) and etomidate (0.3mg/kg) as induction agents. In the study he found out that etomidate group had little changes in mean arterial pressure (MAP) and heart rate when compared to propofol from baseline value. Propofol group had pain on injection during administration and incident of myoclonus were high in etomidate. They concluded that etomidate is better agent for induction than propofol in the view of hemodynamic stability.

Wu J, et al[8] [2013], did a comparison study of anesthetic regimens using etomidate and propofol in patients undergoing first-trimester abortion. A Double-blind, Randomized clinical trial. In this study they compared recovery time, side effects, safety of 6 distinct anesthetic regime for first trimester abortion. 240 women scheduled for surgical abortion from 6 to 8 weeks of gestation were randomized into six groups - (n=40) of propofol: group P (2 mg/kg propofol alone), group PF (2 mg/kg propofol+1 mcg/kg fentanyl), group PMF (2 mg/kg propofol+ 1 mcg/kg fentanyl+0.02 mg/kg midazolam) and (n=40) of etomidate: group E (0.2 mg/kg etomidate alone)group EF (0.2 mg/kg etomidate+

1 mcg/kg fentanyl)group EMF (0.2 mg/kg etomidate+1 mcg/kg fentanyl+0.02 mg/kg midazolam).Vital signs such as pulse oxygen saturation (SpO2), mean arterial pressure (MAP) and heart rate were recorded. The recovery time and side effects were also recorded. They showed that etomidate is much safer in terms of hemodynamics in patient undergoing first trimester abortion. Usage of lower dose of etomidate with fentanyl and midazolam were more beneficial than the use of etomidate with or without fentanyl in avoiding myoclonus and post-operative nausea and vomiting.

Shah Sb [(9)] [2015] did a study in Comparison of hemodynamic effects of intravenous etomidate versus propofol during induction and intubation using entropy guided hypnosis levels. 60 ASA I & II patients in the age group 20-60 years where scheduled for modified radical mastectomy and they were randomly allocated in two groups based on induction agent Etomidate and Propofol. Both groups were premedicated with intravenous midazolam 0.03 mg /kg and fentanyl 2 μg/kg. After induction with the desired agent titrated to entropy 40, vecuronium 0.1 mg /kg was used to administer neuromuscular blockade. Heart rate, systolic, diastolic and mean arterial pressures, response entropy and state entropy were recorded at baseline, induction and upto three minute post intubation. The study concluded that etomidate provided more

hemodynamic stability without the requirements of any rescue drug in 96.6% patient whereas ephedrine was in 36% patient under propofol group

Sanjeeta Umbarkar et al[10] [2015] studied the effects of single induction dose of etomidate on stress response in adult patients undergoing cardiac vascular surgeries with cardiopulmonary bypass .60 patients were randomly allotted in etomidate and fentanyl -midazolam group, 30 in each group. Blood samples for plasma cortisol and blood sugar levels were collected before induction of anaesthesia, after aortic cross clamping on cardio pulmonary bypass and 24 hrs postoperatively. In the study they found that even though etomidate causes adrenocortical suppression it was beneficial in reducing stress response by decreasing cortisol levels and blood sugar levels during intraoperative and postoperative periods. It was also seen that hemodynamic is well maintained in etomidate group compared to high dose opioids, the cortisol and blood sugar levels returns to normal following 24hrs post-surgery, thus single dose of etomidate does not cause much harm by adrenocortical suppression.

Song JC, et al[11] [2015] did a study on etomidate anaesthesia during ERCP caused more stable haemodynamic responses compared

with Propofol: A Randomized Clinical Trial. In his study 80 patients undergoing ERCP were randomly assigned under etomidate or propofol group. Cardiovascular parameters and procedure related time were recorded during ERCP. They found that mean arterial pressure (MAP) values in the etomidate group showed less variation than those in propofol group ($P<0.05$). The recovery time and duration of ERCP was similar in both groups thus they concluded that etomidate anaesthesia during ERCP was hemodynamically stable when compared with propofol.

Kahlon A et al[12][2014], did a randomized double blinded placebo control study on 75 patients. They were divided into 3 groups, 25 patients in each group. Group I received placebo- 1 ml of normal saline, Group II received 1 ml of 2% lignocaine and Grorp III received 1 ml of midazolam (1mg) as premedication 2 mins before induction with etomidate 0.3mg/kg. they observed that incidence of myoclonus in placebo group was 76%, lignocaine group 44% and midazolam group was 28%.

Masoudifar N, et al[13][2013] did a comparison of cardiovascular response to laryngoscopy and tracheal intubation after induction of anaesthesia by propofol and etomidate. In his study 25 consenting ASA I and II patients were randomly divided into two groups, etomidate

(0.3mg/kg) in group A and propofol (2-2.5 mg/kg) in group B and evaluated their cardiovascular responses including systolic blood pressure, diastolic blood pressure, mean arterial pressure and heart rate and SPO2.The above parameters were measured before, during and after laryngoscopy and at 1,3,5,10 min after the intubation. The changes of systolic blood pressure (P=0,019), mean arterial pressure (P=0.008) in the group B was significantly higher than the group A. No significant difference between the groups A and B in terms of heart rate and SPO2. Thus concluding that etomidate is more stable hemodynamically than propofol.

Moller et al[14] [2013] used propofol and etomidate induction of general anaesthesia accompanied by BIS monitoring. 48 patients were studied, the mean arterial pressure , Cardiac index(CI) and systemic vascular resistance index(SVRI) were recorded. The haemodynamic variables were higher in etomidate group upto 7mins after intubation. There was significant hypotension in propofol group. Heart rate and mean arterial pressure was maintained in etomidate group. Incidence of hypertension and tachycardia was more in etomidate group. Hence etomidate was found to be stable compared to propofol in hemodynamic parameters

Tokul S, et al[15] [2009] did a study of comparison of etomidate-remifentanil and propofol -remifentanil sedation in patients scheduled for colonoscopy. In his study 60 patients under the age group of 18 to 65 years where scheduled for elective colonoscopy under sedation. Remifentanil was given in the continuous infusion at the rate of (0.1 mcg/kg/min). After 2 mins propofol (initial dose of 0.5 mg/kg maintained with 0.25 mg/kg) or etomidate (initial dose of 0.1 kg maintained with 0.05 mg/kg were given). Heart rate, mean arterial pressure, SPO_2 and Ramsay sedation score where recorded at baseline, every 2 mins for the first 10 mins and every 5 mins thereafter till the completion of procedure. They found that apnea and hypotension were significantly lower in the etomidate group ($P<0.001$). Thus he concluded that etomidate-Remifentanil combination provided more hemodynamic stability than propofol – remifentanil.

Hildreth AN, et al[16][2008] performed a prospective randomized controlled study. Adrenal suppression following single dose of etomidate for rapid sequence induction. In his study Adult trauma patients were admitted at level I trauma centre who required a rapid sequence induction where randomized to receive etomidate 0.3 mg/kg, succinylcholine 1mg/kg (E group) or fentanyl 100 mcg, midazolam 5mg and succinylcholine 1mg/kg (FM group). Baseline values of serum cortisol

level where measured before rapid sequence induction. A post intubation serum cortisol level where measured at 4 to 6 hours after intubation. An ACTH stimulation test was performed. The use of etomidate for rapid sequence induction in trauma patients, showed the evidence of adrenocortical insufficiency and it had caused increased hospital and ICU stay. Further studies were needed for evaluating the safety of this drug in trauma patients.

Schmidt et al[17] [1999] found in their study that hypotension caused by propofol is due to the reduction of heart's preload and afterload. This hypotension sue to propofol are not synchronized with heart's compensatory responses such as increased cardiac output and increased heart rate. These changes in hemodynamic drop would be intensified by high doses of the drug and high speed injection of the drug. In our study we got similar results in group I which is after induction with propofol there was hypotension which is not synchronized with increased heart rate.

Harris et al[18][1988] did a study comparing the hemodynamic response to tracheal intubation in 303 patients in whom anaesthesia was induced with either thiopentone 4 mg/kg, etomidate 0.3 mg/kg or propofol 2.5 mg/kg with or without fentanyl 2 μg/kg. In patients who

were induced only with propofol had significant decrease in arterial blood pressure the values of which did not increase above baseline value after intubation. There was significant increase in arterial pressure following intubation with thiopentone or etomidate alone. Increase in heart rate occurred with all agents after laryngoscopy and intubation. When fentanyl was added to propofol it resulted in lower arterial pressure on comparing with induction agent alone. We got similar results in our study. Significant decrease in arterial blood pressure occurred with propofol, while with etomidate there was significant increase in arterial pressure following intubation. Also increase in heart rate occurred with all agents after laryngoscopy and intubation.

History of Intravenous Anaesthetics

HISTORY OF INTRAVENOUS ANAESTHETIC DRUGS

William Harvey described the complete and continuous intravascular circuit in 1628. Wren[19] introduced intravenous anaesthesia in 1656 a technique using a goose quill and a bladder to inject wine and ale into a dog's vein. The invention of the hollow needle in 1843 and hypodermic syringe in 1853 allowed IV administration of drugs. In 1900s, diverse drugs including ether had been given for sedation. In 1855, Wood published a paper on the injection of opiates into painful spot by use of hollow needle and a glass syringe.

In 1872, Pierre Ore of Lyons performed first successful surgical anaesthesia by injecting chloral hydrate. In 1909, Ludwig Burkhardt produced surgical anaesthesia by intravenous injection of chloroform and ether in Germany.

First barbiturate was synthesized in 1903 by Fisher and Von Mering. Weese and Scharpff synthesized the short-acting hexobarbital in 1932 but the introduction of thiopental by Lundy and Tovell in 1934 provided greater advancement dominating induction of anaesthesia for the next half century.

Price in 1960 suggested that thiopental redistributes from brain to muscle rather than to fat. This explained rapid awakening following thiopental induction. This work led to discovery of many pharmacokinetic and pharmacodynamics concepts.

Thiopental decreased cerebral metabolism and cerebral blood flow, leading to its use in the 1970s to protect the brain from hypoxic insult. Methohexital, a shorter-acting barbiturate with central nervous system-stimulating properties was introduced in 1956 and it still remains a popular anesthetic for electroconvulsive therapy (ECT).

In 1962, Ketamine was synthesized by Dr. Calvin Stevens. Etomidate was first described by Paul Janssen and his colleagues in 1964. Propofol was first synthesized in 1977 by imperial chemical industries. Cremophor EL, the solvent which was formulated produced severe allergic reactions and was withdrawn from usage. Then propofol was formulated with egg lecithin, soya bean oil, glycerol, it entered practice and grained more importance.

Stress Response and Anaethesia

STRESS RESPONSE AND ANAESTHESIA

The body reacts to external stimuli, minor to major both locally and generally leading to widespread endocrinal, metabolic, and biochemical reactions throughout the whole body. So amplitude of the response depends upon severity, intensity and duration of stimulus. The complex interplay of substances following the trigger between the hypo thalamo pituitary axis and classical neuro endocrine hormone system and autonomic nervous system results in a reaction called stress response or alarm reaction.

The stress response leads to secretion of various anabolic and catabolic hormones resulting in hypermetabolic state. In acute phase this helps in maintaining hemodynamic state. If it is prolonged, the continuous hypermetabolic state results in exhaustion of essential components of the body leading to decreased resistance, increased mortality and morbidity. The net effect of stress response "The Neuroendocrine outflow":

- Cardiovascular changes: - Rise in cardiac output, heart rate, blood pressure, increased myocardial contractility, increase oxygen demand.

- ❖ Blood volume distribution: – Peripheral and splanchnic vasoconstriction coronary and cerebral vasodilatation.
- ❖ Respiratory Changes- Increased respiratory rate.
- ❖ Fluid and electrolyte changes: - Sodium and water retention.
- ❖ Coagulation: - Hyper coagulability and fibrinolysis.
- ❖ Immunosuppression: - Wound Infections.
- ❖ Metabolic Changes: – Substrate mobilization hyperglycemia.
- ❖ Urinary changes: – reduced urinary output.

The stress changes are well tolerated in ASA grade I and II patients. In patients with co-morbidities these changes leads to life threatening complications.

The primary stimuli of neuro-endocrine reflexes

1. Hypotension
2. Oxygen, carbon dioxide and hydrogen ion imbalance
3. Anxiety and emotional disturbances
4. Temperature variations
5. Anaesthesia

- Anesthetic drugs
- Laryngoscopy and intubation

Following the mechanical stimulation of upper airway tract, efferents that are carried by glossopharyngeal nerve and via vagus nerve from tacheo-broncheal tree enhances the activity of cervical sympathetic afferent fibres resulting in transient increase in heart rate and blood pressure.

- Light plane of anaesthesia
- Pain

6. Surgery

The stress response is divided into two phases:

- Acute or shock phase.
- Slow phase.

The stress hormones are

♣ Autocrines: Catecholamines, Glucagon, Insulin.

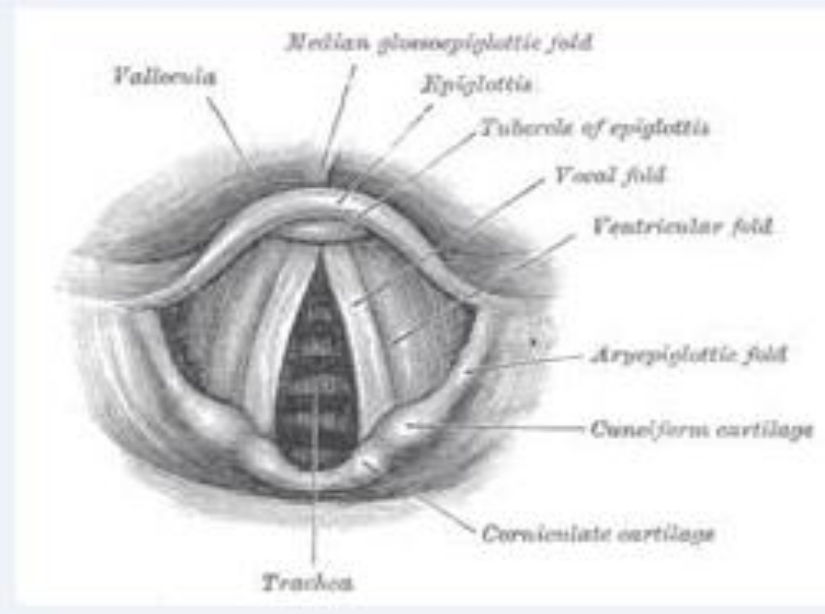
♣ Endocrines: Cortisol, Growth hormone, Vasopressin, Aldosterone, Renin-Angiotensin.

♣ Paracrines: Cytokines, Leukotrienes etc.

Physiological changes during laryngoscopy

PHYSIOLOGICAL CHANGES DURING LARYNGOSCOPY

The airway manipulations like laryngoscopy and endotracheal intubation are noxious stimuli. These external stimuli may produce profound changes in cardiovascular physiology primarily through reflex responses. Even though these responses are of short duration and causes minor consequence in healthy individuals, serious complications occur in those patients with underlying coronary artery disease, reactive airways or intracranial neuropathology.



Cardiovascular Responses during Airway Manipulation

Cardiovascular Reflexes

The cardiovascular response[20]to external and noxious airway manipulation are initiated by proprioceptors responding to tissue irritation in the supraglottic region and in the trachea. These receptors are located in close proximity to the airway mucosa, it consists of mechanoreceptors with small-diameter myelinated fibers. These fibers are slowly-adapting stretch receptors, with large-diameter myelinated fibers and polymodal endings of nonmyelinated nerve fibers. Autonomic activation is caused by glossopharyngeal and vagal afferent nerves that transmit these impulses to the brainstem there by activating sympathetic and parasympathetic nervous systems.

Hypertension and tachycardia mediated by the cardio accelerator nerves and sympathetic chain ganglia are the most common response to airway manipulation in adults and adolescents. This response causes widespread release of norepinephrine from adrenergic nerve terminals. There is secretion of epinephrine from the adrenal medulla. Renin-angiotensin system is also activated by the hypertensive responses to endotracheal intubation leading to release of renin from the renal juxtaglomerular apparatus which is innervated by β-adrenergic nerve terminals.

Laryngoscopy and endotracheal intubation result in stimulation of the central nervous system seen by increases in electroencephalographic activity, cerebral metabolic rate, and cerebral blood flow.

Acute bronchospasm or a main stem bronchial intubation in a disturbed perfusion to poorly ventilated lung units causing desaturation of pulmonary venous blood. This events results in reduction in systemic arterial oxygen tension. In addition, institution of positive end expiratory pressure after endotracheal intubation causes a reduction in cardiac output mediated by decreased venous return to the left side of the heart from the pulmonary circulation. The impact of these changes can be profound in patients with severely compromised myocardial function and in patients with intravascular volume depletion.

Intubation in the Presence of Cardiovascular Disease

Myocardial ischemia occur when there is an imbalance between myocardial O2 supply and demand. Heart rate and myocardial wall tension are the two components that determine the myocardial oxygen demand.. Of the two increases in heart rate are of greatest concern because cardiac inotropism subserves cardiac chronotropism rate. Not only tachycardia increases myocardial O2 consumption per minute at

constant wall tension. But this changes in tachycardia causes decrease in diastolic period.

Patients with aneurysmal disease of the cerebral and aortic circulation are at risk of complications related to a sudden increase in BP during airway instrumentation. Laplace's law defines the transmural wall tension of a blood vessel as the product of the pressure inside the vessel and its radius divided by the wall thickness.

Implications for Patients with Neurovascular Disease

In patients with intracranial aneurysms and arteriovenous malformations, periods of elevated arterial blood pressure causes these lesions to rupture resulting in sudden and permanent neurologic injury. Therefore, neurosurgical anesthesiologists must pay careful attention to reducing these responses during the course of anesthetic induction and endotracheal intubation.

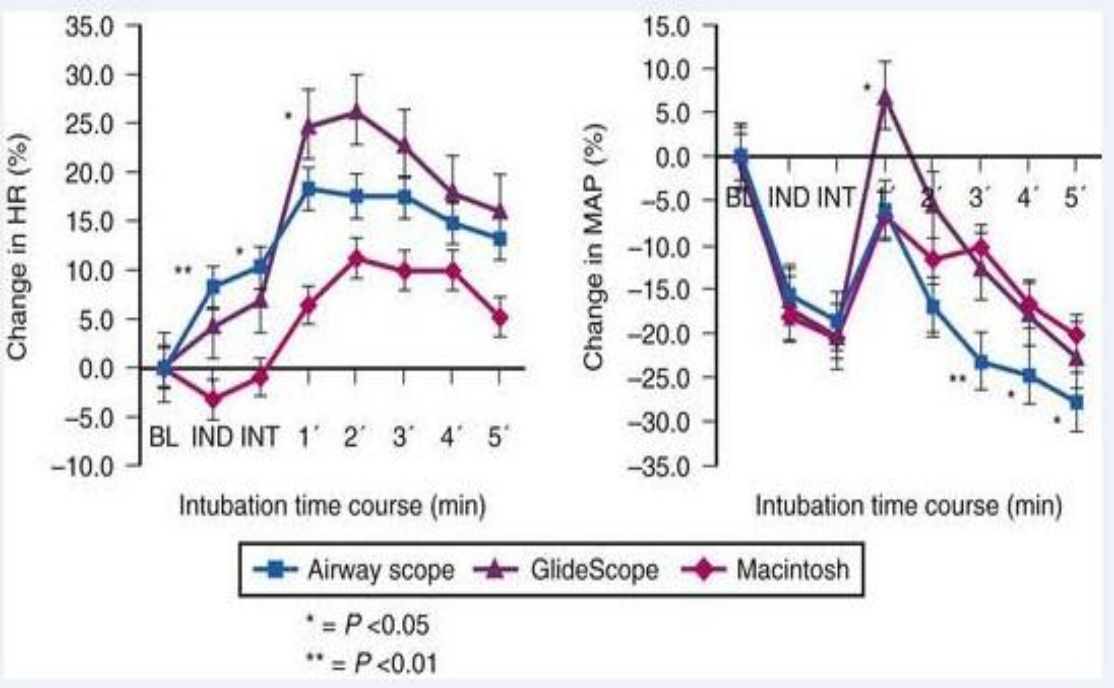
Intubation in Patients with Neuropathological Disorders

The cerebral blood flow (CBF) remain constant over the BP range of 50 to 150 mm Hg. This range is impaired when there is impaired cerebral autoregulation. When endotracheal intubation causes an increase in arterial blood pressure there is a marked increase in CBF and cerebral

blood volume which leads to increases in intra cranial pressure that can be dangerous.

Prevention of Cardiovascular Responses

Technical Consideration: Minimizing Stimulation of Airway. Cardiovascular responses to airway manipulations can be reduced by limiting airway proprioceptor stimulation starting from the manipulation of the larynx itself.



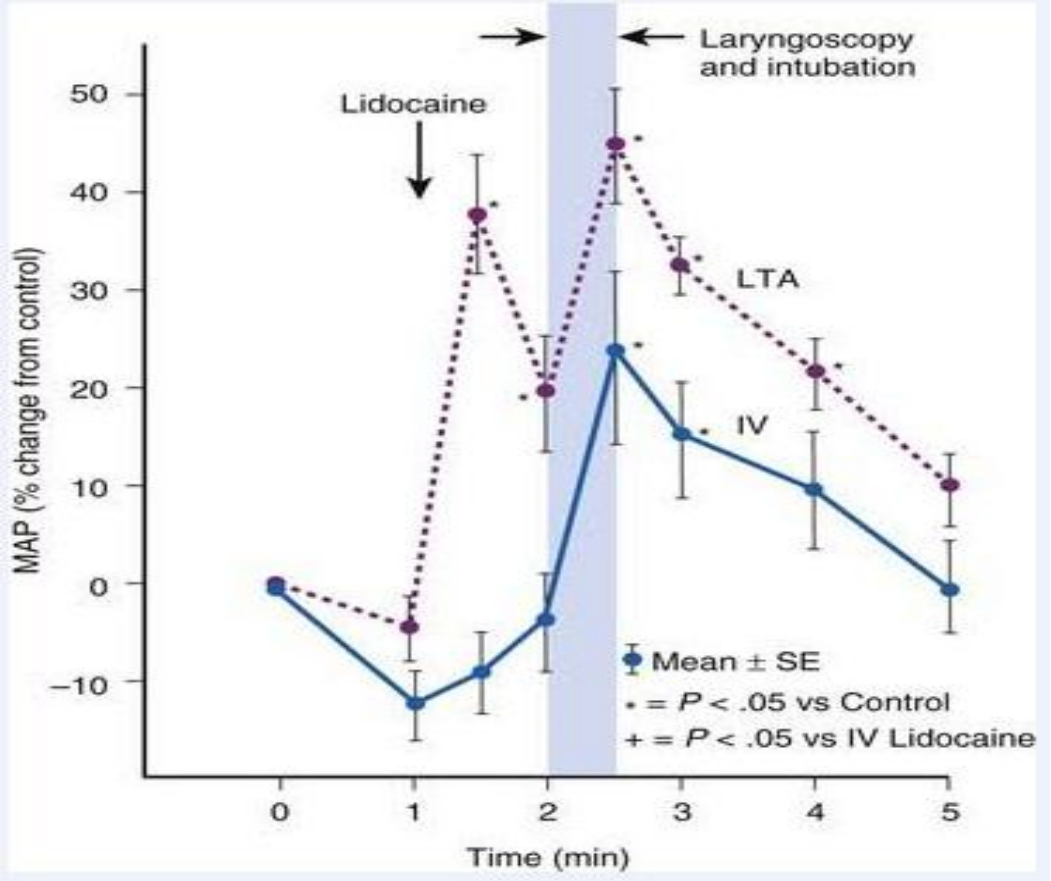
Laryngoscopy [21] itself is a moderately stimulating procedure. Use of a straight blade for elevation of the vaguely innervated posterior aspect of the epiglottis results in significant raise in arterial blood pressure than use of McCoy blade or other newer blades. The above chart shows the

effect of various laryngoscopes in hemodynamic changes to laryngoscopy.

The act of passing an endotracheal tube is far more hemodynamically stimulating than just laryngoscopy. Insertion of a conventional laryngeal mask airway after induction of general anaesthesia with thiopental or propofol and fentanyl, cause less cardiovascular and endocrine response than laryngoscopy or endotracheal intubation. Topical anaesthesia[22] when applied to the upper airway is effective in blunting hemodynamic responses to endotracheal intubation.

In contrast to topical anaesthesia of the airway, which provide inconsistent benefits, regional nerve blocks that involves the sensory pathways from the airway prevent hemodynamic responses to intubation. The superior laryngeal nerve innervates the superior surface of the larynx and the glossopharyngeal nerve innervates the oropharynx. Depositing local anesthetic on each cornua of the hyoid bone can block the superior laryngeal nerve (SLN).

The glossopharyngeal nerve blocked at the tonsillar pillars potentiates this effect by decreasing the stimulus of laryngoscopy.



The above chart shows the effect of lidocaine in attenuation of hemodynamic response to laryngoscopy.

Intravenous Agents

The anesthetic dose of intravenous anesthetic required for effectively blocking hemodynamic and intra cranial response responses to endotracheal intubation has remained an elusive goal.

Propofol, barbiturates, and benzodiazepines are all associated with profound fall in blood pressure at doses that suppress the hemodynamic and intra cranial pressure (ICP) responses to intubation. The effective dose of etomidate for blocking this cardiovascular response to intubation can be identified by a burst-suppression pattern on EEG, which shows fairly deep cerebral depression. It is probably the only agent that by itself can achieve suppression of cardiovascular responses at a dose that does not cause undue arterial hypotension and compromise of coronary and cerebral perfusion.

It is clinically impractical to achieve sufficient anesthetic depth for preventing a hemodynamic response to intubation solely with an intravenous or inhalational agent a wide variety of anaesthetic drug combinations, adjuvants, or both have been used to potentiate anesthetic effects while minimizing hemodynamic depression.

Opioids are the adjuvants most commonly added to iv or inhaled agents to facilitate induction of anaesthesia and subsequent airway manipulation. Fentanyl is 100 times more potent than morphine. Fentanyl is not a short-acting agent, and the risk of prolonged postoperative respiratory depression following fentanyl must be kept in mind against the advantages of perioperative cardiovascular stability. With this risk in

mind, it is seen that pre-treatment with 2 µg/kg IV fentanyl, when given 10 minutes before intubation during an infusion of propofol sufficient to reduce the Bispectral Index Score to 45, thus preventing a significant increase in heartrate and blood pressure compared with awake pre anaesthetic values. Opioids with shorter onset and offset times have much added advantages over fentanyl for modulating circulatory responses to intubation. IV lidocaine may also blunt hemodynamic and cerebrovascular responses to intubation. When given in a bolus of 1.5 mg/kg IV, it adds 0.3 MAC of anaesthetic potency.

Inhalational Anesthetics

For inhalational anesthetics, endotracheal intubation in the dose range of the minimum alveolar concentration resulted in marked cardiovascular stimulation during anaesthesia.

Accordingly, a high minimum alveolar concentration of inhaled anaesthetic drugs is required to block the cardiovascular response to endotracheal intubation. This causes profound cardiovascular depression before endotracheal intubation.

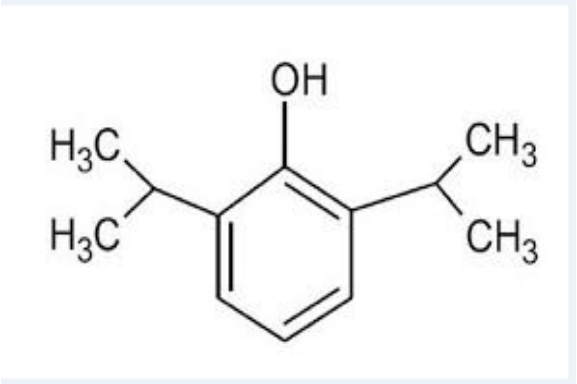
Pharmacology of Propofol

PHARMCOLOGY OF PROPOFOL

PROPOFOL

Propofol is an intravenous sedative-hypnotic that was introduced for clinical use in 1977. It is famously called as “MILK OF AMNESIA”

CHEMICAL STRUCTURE



Propofol is a substituted isopropylphenol (2,6-diisopropylphenol) that is administered intravenously as 1 % solution in an aqueous solution of 10% soybean oil, 2.25% glycerol, and 1.2% purified egg phosphatide Administration of propofol, 1.5 to 2.5 mg/kg IV (equivalent to thiopental, 4 to 5 mg/kg IV, or methohexital, 1.5 mg/kg IV) as a rapid IV injection produces unconsciousness within about 30 seconds.

PREPARATIONS

DIPRIVAN (1%)

Current formulations of propofol use a 10% soybean oil as the oil phase and 1.2% egg phosphatide as the emulsifying agent that is composed of long-chain triglycerides 2.25% glycerol. The generic formulation of propofol incorporates sodium metabisulfite (0.25 mg/mL) or 0.005% sodium edenate as the preservative and has a lower pH (4.5 to 6.4). Propofol, unlike thiopental, etomidate and ketamine, is not a chiral compound.

AMPOFOL

More recently a lower lipid formulation of propofol has been introduced into clinical practice.it contains 5%soyabean oil, 0.6% egg lecithin.

The increased “free” fraction of propofol leads to increased pain when injected into small veins. Therefore it is important to add lidocaine to the Ampofol formulation to minimize the pain on injection.

AQUAVAN (FOSPROPOFOL)

A new water soluble pro drug of propofol. This prodrug is rapidly hydrolyzed by plasma alkaline phosphatases in the circulation to release free propofol. It has slower onset but a similar recovery profile but does not cause pain on injection. A transient burning sensation has been reported following IV injection.

OTHERS

Diprifusor TCI devices , Propofol lipura

MECHANISM OF ACTION

MECHANISM OF ACTION

GABA RECEPTOR COMPLEX

Propofol is relatively selective modulator of GABA receptors

g- amino butyric acid is inhibitory neurotransmitter in brain.

Assemble to form cl- channel with GABA A receptor

Activation of GABA receptors → increases transmembrane CL conductance

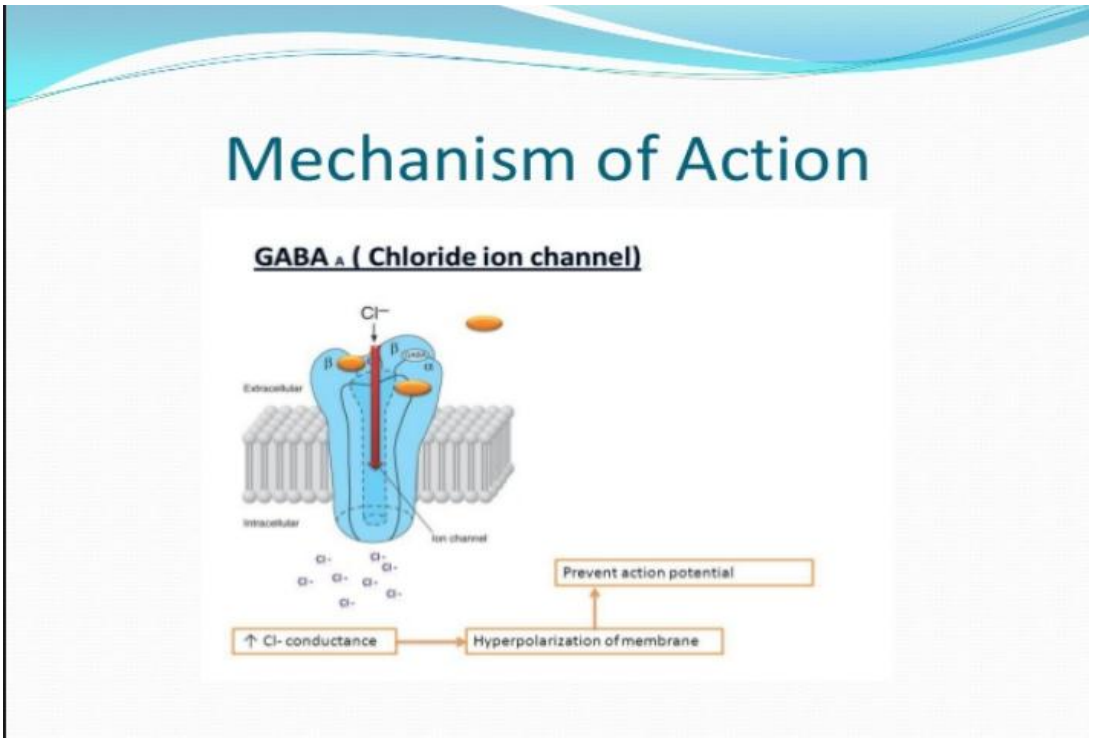
↓

hyperpolarization of post synaptic membrane

↓

functional inhibition of the postsynaptic neuron

Propofol decreases the rate of dissociation of GABA from its receptors, thereby increasing the duration of the GABA –activated opening of chloride channel with resulting hyperpolarisation of cell membrane.



It is a selective modulator of gamma amino butyric acid ($GABA_A$) receptors[23] and it does modulate other ligand-gated ion channels at clinically used concentrations. Propofol exerts its sedative-hypnotic effects through a $GABA_A$ receptor interaction. GABA is the principal inhibitory neurotransmitter in the CNS. On activation of $GABA_A$ receptors, transmembrane chloride conductance increases, There is hyperpolarization of the postsynaptic cell membrane and this leads to functional inhibition of the postsynaptic neuron. The interaction of propofol (also etomidate and barbiturates) with specific components of $GABA_A$ receptors decreases the rate of dissociation of GABA from the receptor, leading to increase in the duration of the GABA-activated opening of the chloride channel which results in hyperpolarization of cell membranes. In similarity to volatile anesthetics, spinal motor neuron excitability, as measured by H-reflexes, is not transformed by propofol, suggesting that immobility during propofol anaesthesia is not caused by drug-induced spinal cord depression.

Widespread inhibition of NMDA (N-Methyl –D-aspartate) receptor through sodium channel gating. Acts on $GABA_A$ and glycine receptors and dorsal horn of spinal cord and inhibits the excitatory action. Increases the Dopamine concentration in nucleus accumbens which is responsible

for sense of wellbeing, drug abuse potential and pressure seeking behavior.

Decreases serotonin levels in area postrema. This causes the antiemetic effect.

Pharmacokinetics

Elimination Half-Time (hrs)	-	0.5-1.5
Volume of Distribution (liters/kg)	-	3.5-4.5
Clearance (mL/kg/min)	-	30-60

Metabolism

Propofol metabolism in humans is both hepatic and extra hepatic. Hepatic metabolism is rapid and extensive, resulting in inactive, water-soluble sulfate and glucuronic acid metabolites. These metabolites are excreted by the kidneys. Propofol undergoes ring hydroxylation by the enzyme cytochrome P-450 to form 4-hydroxypropofol which is then glucuronidated or sulfated.

Clinical use

Propofol is the induction drug of choice for many forms of anaesthesia, especially when rapid and complete awakening is required.

For induction

The induction dose of propofol in healthy adults is 1.5 to 2.5 mg/kg IV, with blood levels of 2 to 6 mg/mL which produces unconsciousness depending on related medications and the patient's age

For intravenous sedation

The conscious sedation dose of 25 to 100 mcg/kg/minute IV. At this dose it produces minimal analgesic and amnestic effects.

For maintenance of anaesthesia

The typical dose of propofol for maintenance of anaesthesia is 100 to 300 mcg/kg/minute, often used in mixture with a short-acting opioid.

Nonhypnotic Therapeutic applications :

- For Anti-emetic effect.
- For Anti-pruritic effect.
- For Anti-convulsant activity.
- For attenuation of bronchoconstriction.

Effects on other systems

Central Nervous System

Propofol causes a decrease in cerebral metabolic rate, cerebral blood flow and intracranial pressure. The change in PaCo2 is similar to change in cerebral blood flow velocity. Propofol produces cortical electroencephalographic (EEG) changes that are similar to those of thiopental, At higher doses it produce burst suppression of cortical somatosensory evoked potentials which is used to monitoring spinal cord function. These functions are not significantly modified in the presence of propofol alone, but on addition of nitrous oxide or a volatile anesthetic results in decreased amplitude.

Cardio vascular system

Propofol produces decreases in systemic blood pressure[(24)] that are greater than thiopentone . The decrease in blood pressure is due to changes in cardiac output and systemic vascular resistance. Inhibition of sympathetic vasoconstrictor nerve activity by propofol causes relaxation of vascular smooth muscle. A negative inotropic effect of propofol, is due to secondary to inhibition of trans-sarcolemmal calcium influx which results in decrease of intracellular calcium availability for cardiac muscles to contract. Stimulation produced by direct laryngoscopy and

intubation of the trachea reverses the blood pressure effects of propofol. It is more effective than thiopental in blunting this presser response.

Respiratory System

Apnea occurs in 25% to 35% of patients after induction of anaesthesia with propofol, this is due to dose dependent depression of ventilation. It also depresses the central chemoreceptors which is the reason for decreased response to hypercarbia. It causes bronchodilation and decrease the incidence of intraoperative wheezing in patients with asthma.

Hepatic and Renal Function

Propofol has no effect hepatic or renal function as reflected by measurements of liver transaminase enzymes or creatinine concentrations. On prolonged infusions of propofol, there are cases associated with hepatocellular injury accompanied by lactic acidosis, rhabdomyolysis and arrhythmias.

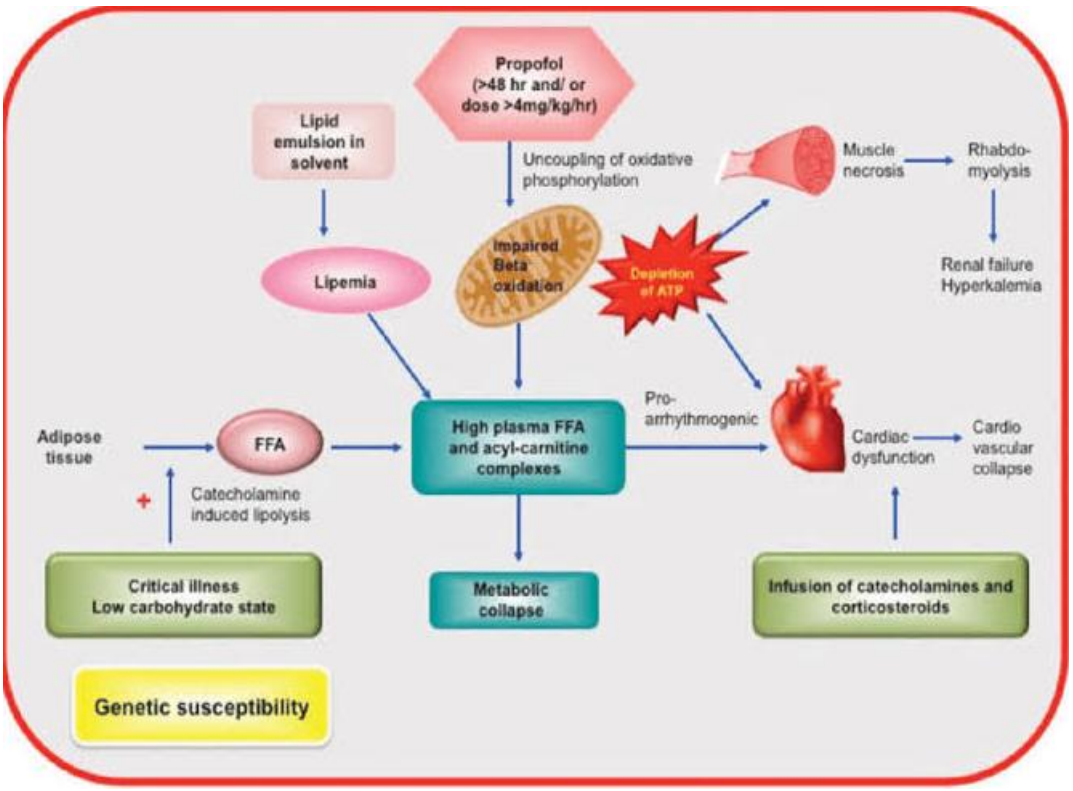
Coagulation:

Inhibits platelet aggregation, by proinflammation lipid mediators that includes thromboxane A2 and platelet-activating factor.

Side effects

- Allergic reactions
- Lactic acidosis
- Pro convulsant activity
- Abuse potential
- Pain on injection.

PROPOFOL INFUSION SYNDROME



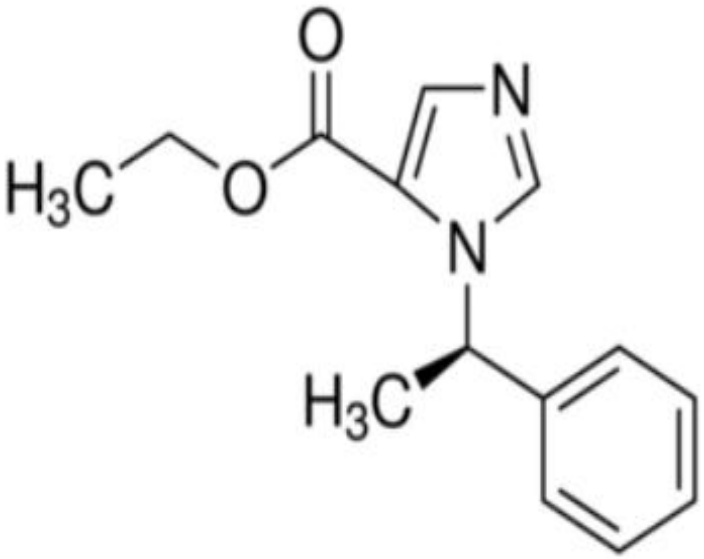
Propofol infusion syndrome (PRIS) is a rare syndrome that occurs in patients, who are on prolonged propofol infusion for sedation.it occurs when more than 4mg/kg/hr of propofol is infused for more than 24 hrs. It

is associated with cardiac failure, rhabdomyolysis, metabolic acidosis, and kidney failure, which are often fatal. High blood potassium, high blood triglycerides, and liver enlargement is due to direct mitochondrial respiratory chain inhibition or impaired mitochondrial fatty acid metabolism. It is common with children, and critically ill patients and those whose are receiving catecholamines and glucocorticoids. Early recognition of this syndrome is very important and discontinuation of the propofol infusion decreases morbidity and mortality. Treatment is supportive measures.

Pharmacology of Etomidate

PHARMOCOLOGYOF ETOMIDATE

ETOMIDATE



Etomidate, carboxylated imidazole-containing compound. The imidazole nucleus of the etomidate renders it water soluble at an acidic pH and lipid soluble at physiologic pH.

COMMERCIAL PREPARATION

- With propylene glycol 35%
- Fat emulsion
- Oral preparation for transmucosal delivery
- Acidic pH 6.9 pKa 4.2, poorly water soluble

MECHANISM OF ACTION

Etomidate [25] is a relatively selective as a modulator of GABA receptors. It exerts its effect by binding directly to a specific site or sites on the protein of $GABA_A$ receptors and enhancing the affinity of the inhibitory neurotransmitter.

PHARMACOKINETICS

The volume of distribution (Vd) of etomidate is large, leading to considerably large tissue uptake. Etomidate reaches the brain rapidly, reaching peak levels within 1 minute after IV injection. The redistribution of the drug from brain to inactive tissue sites leads to rapid awakening following single dose of etomidate. Rapid metabolism is also adds to its prompt recovery

METABOLISM

Hydrolysis of the ethyl ester side chain to its carboxylic acid ester, results in a water-soluble and pharmacologically inactive compound. Hydrolysis is nearly complete, < 3% of an administered dose of etomidate excreted as unchanged drug in urine. The clearance of etomidate is about five times of thiopental; this reflect shorter elimination half-time of 2 to 5 hours.

CLINICAL USES

Etomidate can be used as an alternative to propofol or barbiturates for the IV induction of anaesthesia, especially when there is unstable cardiovascular system. Standard induction dose of etomidate is 0.2 to 0.4 mg/kg. The onset of unconsciousness occurs within one arm-to-brain

circulation time. Involuntary myoclonic movements are common during the induction.

The limiting for clinical use of etomidate following induction of anaesthesia is the ability of this drug to transiently depress adrenocortical function.

SIDE EFFECTS

Central Nervous System

Potent direct cerebral vasoconstrictor that decreases cerebral blood flow and CMR02, 35% to 45%, etomidate produces a pattern on the EEG that is similar to thiopental. The frequency of excitatory spikes on the EEG is greater with etomidate than with thiopental and methohexital, must be cautiously used in patients with seizure disorder.

Cardio Vascular System

At 0.3 mg/kg IV of etomidate there are minimal changes in heart rate, stroke volume, or cardiac output. The mean arterial blood pressure may decrease up to 15% due to decreases in systemic vascular resistance. The decrease in systemic blood pressure is similar to changes in systemic

vascular resistance. This suggests that administration of etomidate to acutely hypotensive patients could result in sudden hypotension.

At induction dose of etomidate 0.45 mg/kg there is decrease in systemic blood pressure and cardiac output. The cardiovascular effects of etomidate and thiopental are comparable when given continuously to patients with severe valvular heart disease.

VENTILATION

The depressive effects of etomidate on ventilation seems to be less significant than barbiturates, although apnea may occasionally occur on a rapid IV injection of the drug.

PAIN DURING INJECTION

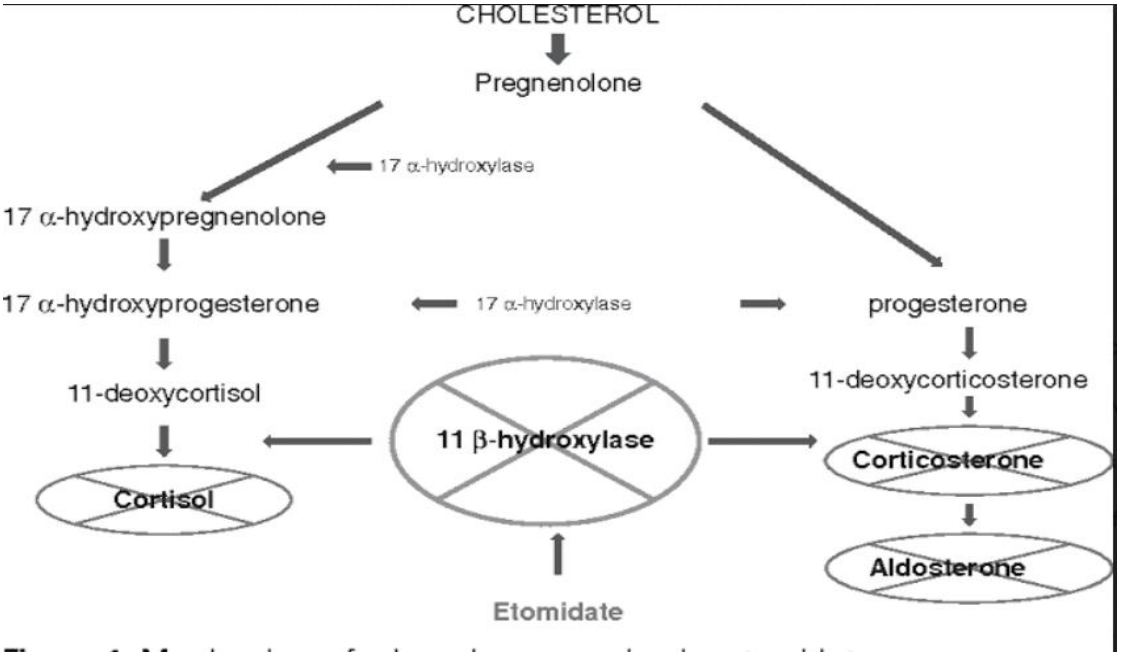
With the use of lipid emulsion vehicle, Pain on injection and venous irritation has been virtually eliminated.

MYOCLONUS

The mechanism of etomidate-induced myoclonus is due to disinhibition of subcortical structures which suppress extrapyramidal motor activity. In many patients with excitatory movements, EEG shows early slow phase which corresponds to the beginning of deep anaesthesia.

ADRENO CORTICAL SUPPRESSION[26]

Inhibition of the conversion of cholesterol to cortisol by etomidate causes adrenocortical suppression. The mechanism of which is due to dose-dependent inhibition of the conversion of cholesterol to cortisol. The particular enzyme inhibited by etomidate appears to be 11-beta-hydroxylase as shown by the accumulation of II-deoxycorticosterone. This enzyme inhibition lasts 4 to 8 hours after an induction dose of etomidate.



ALLERGIC REACTIONS

The incidence of allergic reactions following administration of etomidate is very low with low flows for prolonged periods.

Materials and Methods

MATERIALS AND METHODS

STUDY CENTRE

ESIC MEDICAL COLLEGE AND PGIMSR, KK NAGAR, CHENNAI- 78.

After institutional ethical committee approval, study was conducted in sixty patients between 20-50 years of age, of either sex, weighing 40 to 80 kg and ASA grade I or II scheduled for elective surgery under general anaesthesia after obtaining informed consent from the patients.

DURATION OF THE STUDY

Jan 2017 – May 2018

STUDY DESIGN

Randomized double blinded controlled interventional study

METHODS

After institutional ethical committee approval, study was conducted in sixty patients between 20-50 years of age, of either sex, weighing 40 to 80 kg and ASA grade I or II scheduled for elective surgery under general anaesthesia after obtaining informed consent from the patients.

SAMPLE SIZE

Based on a previous study by Masoudifar et al, it was seen that patients who received propofol (26%) had hypotension following intubation compared to etomidate (8%). Based on this study, the sample size was calculated using n Master 2.0 software with an alpha error of 5% and power of 80%. Sample size was found to be 25 per group and rounded off to 30 per group to account for drop outs.

ANALYSIS PLAN

Collected data were analysed using statistical package SPSS version 21.0

INCLUSION CRITERIA

- 20 – 50 years of either sex
- Weight 40 to 80 kgs.
- ASA grade I and II.
- Mallampati grade I and II.

EXCLUSION CRITERIA

- Patient refusal.
- Emergency surgeries.

- Patients with cardiovascular diseases like Ischemic heart disease(IHD) and hypertension.
- Existence of considerable pathology in pharynx/larynx.
- Patients on beta blockers and antihypertensive medication.
- Diabetic patients.
- Known history of allergy to propofol or etomidate.
- History of seizure disorder

PREOPERATIVE PREPARATION

All the patients were subjected to basic haematological and biochemical investigations which included hemoglobin, total count, differential count, platelet count, renal and liver function tests, random blood sugar. 12 lead ECG, chest x ray and USG abdomen were also taken for all the patients.

The patients were kept nil per oral for 8 hours before surgery. All the patients were given tablet Alprazolam 0.25 mg, tab Ranitidine 150 mg and tab Metoclopramide 10 mg on the night before the surgery. Tab ranitidine 150 mg and tab metoclopramide 10 mg on the morning of surgery given at 6 am with sips of water

On arrival to the preoperative room, informed consent was obtained for the participation of the patient in the study. The patients were randomly allocated into two groups by sealed envelope technique into:

GROUP A–Propofol 30 patients

GROUP B- Etomidate 30 patients

Patient was shifted to the operation theatre by trained personnel on a trolley.

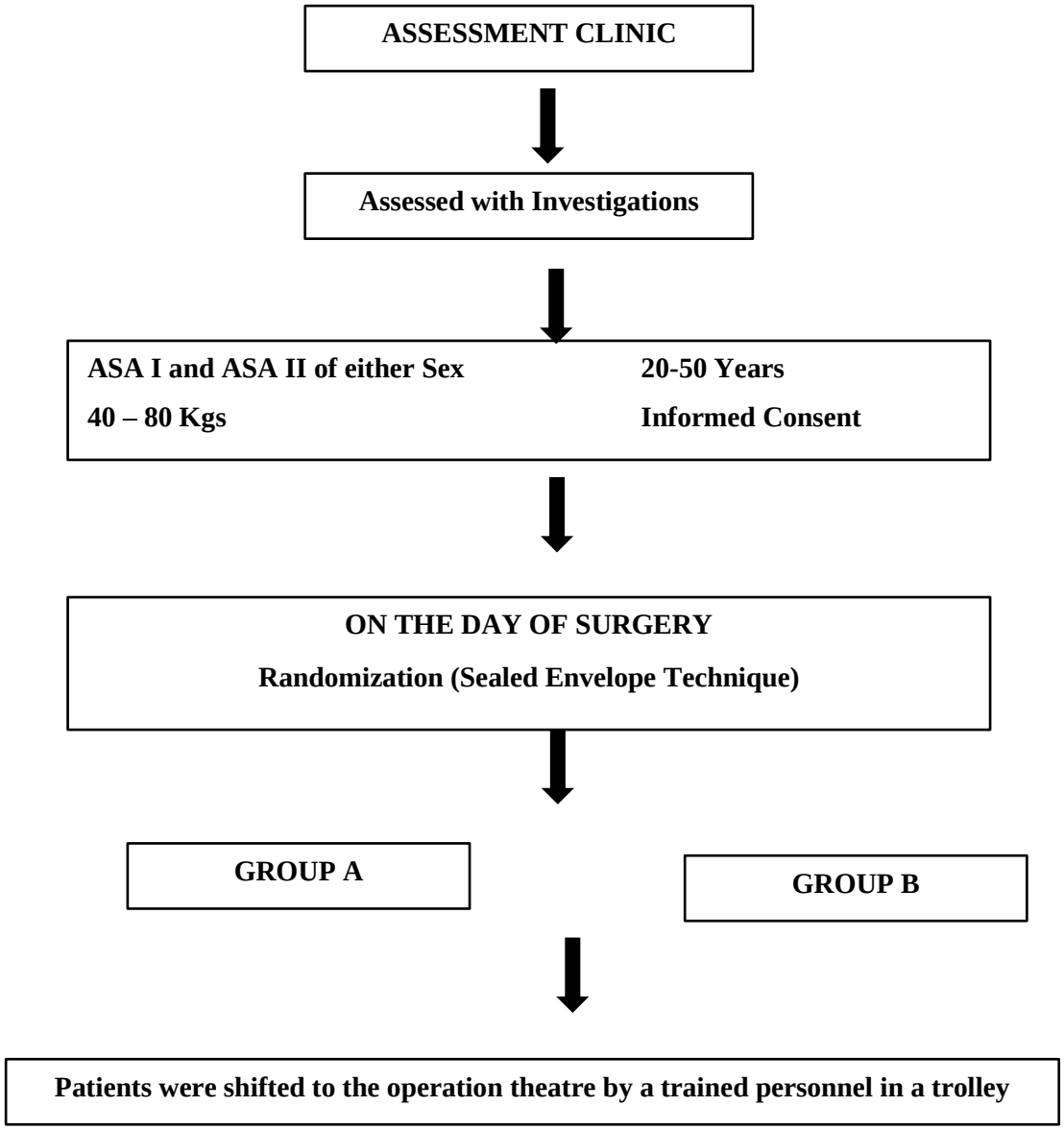
PROCEDURE DETAILS

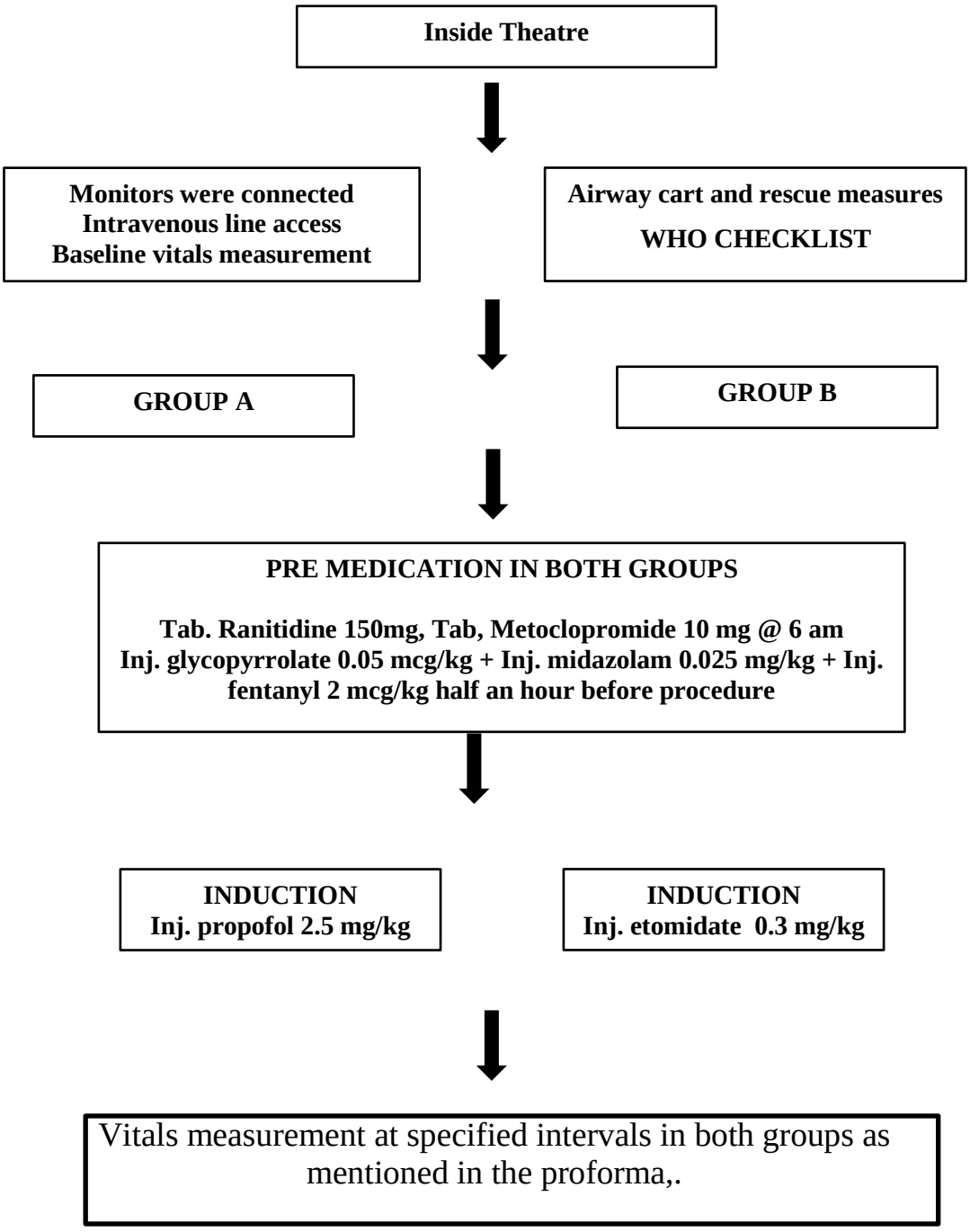
- WHO CHECKLIST was performed for all the patients.
- In operation theatre, standard monitors - Pulse oximetry for saturation (SpO2), Noninvasive blood pressure monitoring (NIBP), Electrocardiogram (ECG), ETCO2 were connected and baseline pulse rate, mean arterial pressure, ETCO2 and oxygen saturation were recorded. Intravenous line access obtained with 18 G cannula and crystalloid infusion started. Airway cart and rescue measures were kept ready.
- Premedication: Injection midazolam 0.025mg/kg IV, injection glycopyrrolate 0.05mcg/kg IV and injection fentanyl 2µg/kg IV. Hemodynamic variables were noted
- Preoxygenation was done with 100% oxygen for 5 minutes
- For Induction Group A patients received Inj Propofol 2.5 mg/kg IV
- For Induction Group B patients received Inj Etomidate 0.3mg/kg IV
- All the drugs that were used in the study were prepared by myself. It was decided that if any complication or untoward incidence

arise, blinding will be unfolded, patients will be treated accordingly. Drug given by anaesthesiologist who were blinded during the study. Speed of injection (10 secs) were equal in both the groups. After induction of anaesthesia, hemodynamic variables were recorded.

After loss of consciousness, which was confirmed by inability to respond to verbal commands and loss of eyelash reflex, Inj. vecuronium (0.08 mg/kg) was given; patient was ventilated with oxygen: nitrous oxide @ 33% : 66% and Isoflurane 1% dial concentration. Laryngoscopy and endotracheal intubation was done by experienced anesthesiologist who is blinded after 5 minutes. Duration of laryngoscopy was kept less than 10 seconds. Cases were excluded when laryngoscopy time more than 10 seconds or in patient with unanticipated difficult airway and was proceeded according to difficult airway algorithm. Trachea was intubated with adequate size endotracheal tube. Proper placement of endotracheal tube was confirmed by capnography and 5 point auscultation of chest. Following successful placement of ET tube anaesthesia was maintained with isoflurane 1-1.5% dial concentration and oxygen-nitrous oxide at the ratio of 33%:66%. Vitals were recorded at induction, post induction, 1, 2, 3,5 and 10 minutes after intubation. Later, the anaesthesia was maintained with standard protocol.

PATIENT FLOW CHART




Inside Theatre
Monitors were connected
Intravenous line access
Baseline vitals measurement
Airway cart and rescue measures
WHO CHECKLIST
GROUP A
GROUP B
PRE MEDICATION IN BOTH GROUPS
Tab. Ranitidine 150mg, Tab, Metoclopromide 10 mg @ 6 am
Inj. glycopyrrolate 0.05 mcg/kg + Inj. midazolam 0.025 mg/kg + Inj. fentanyl 2 mcg/kg half an hour before procedure
INDUCTION
Inj. propofol 2.5 mg/kg
INDUCTION
Inj. etomidate 0.3 mg/kg
Vitals measurement at specified intervals in both groups as mentioned in the proforma,.

Observation and Results

OBSERVATION AND RESULTS

The data collected was analyzed using SPSS software version 21 (statistical package for social science). Continuous variables were given by means with standard deviation. Categorical variables were given by frequency and percentages. Student t-test was used for testing the significance of all the variables, means and standard deviation. Chi – square test was used to compare the proportions. All the statistical results were considered significant at the p value of less than 0.05.

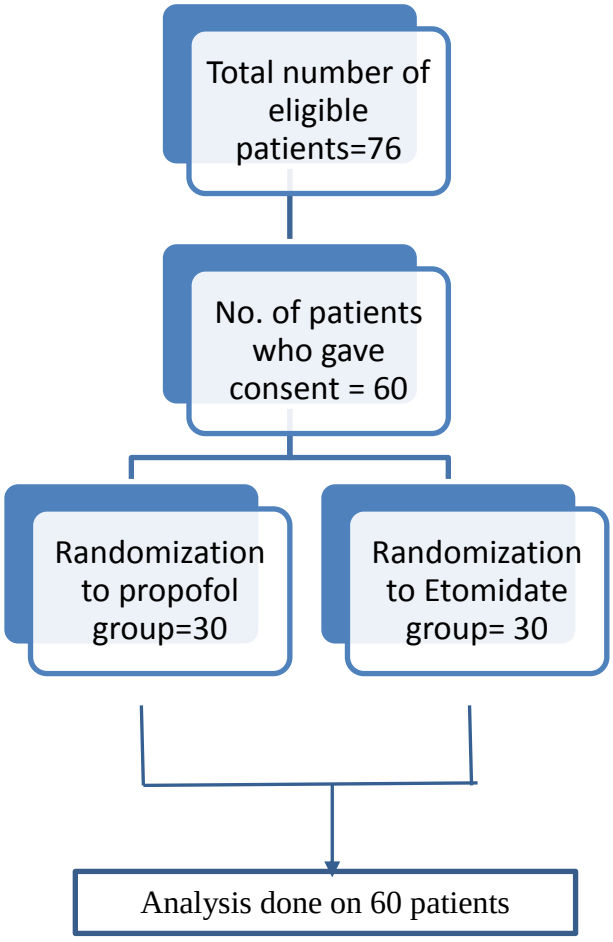


CHART-1

GENDER DISTRIBUTION

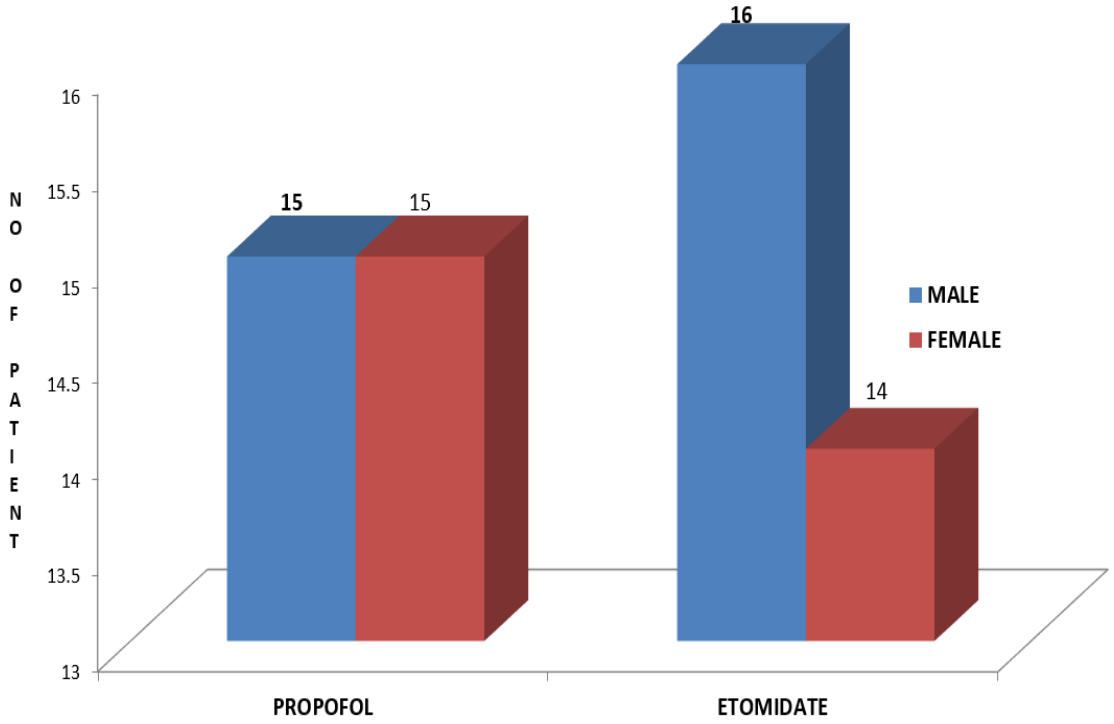


TABLE-1

GENDER DISTRIBUTION

	PROPOFOL		**ETOMIDATE**		**TOTAL**	
	NO	**%**	**NO**	**%**	**NO**	**%**
MALE	15	50.00	16	53.33	31	51.67
FEMALE	15	50.00	14	46.67	29	48.33
TOTAL	30	100	30	100	60	100
SEX RATIO (Male: Female)	**15 : 15**		**16 : 14**		**31 : 29**	
Chi square value	0.07					
df	58					
p-value	0.80* **(Not Significant)**					

*P value >0.05 Not Significant

Propofol and Etomidate groups were comparable with respect to gender distribution. Male and female were more or less equally distributed in both the groups. There were 15 males and 15 females in the propofol group and 16 males and 14 females in the etomidate group. The P value was 0.08 therefore statistically not significant.

CHART-2

AGE DISTRIBUTION

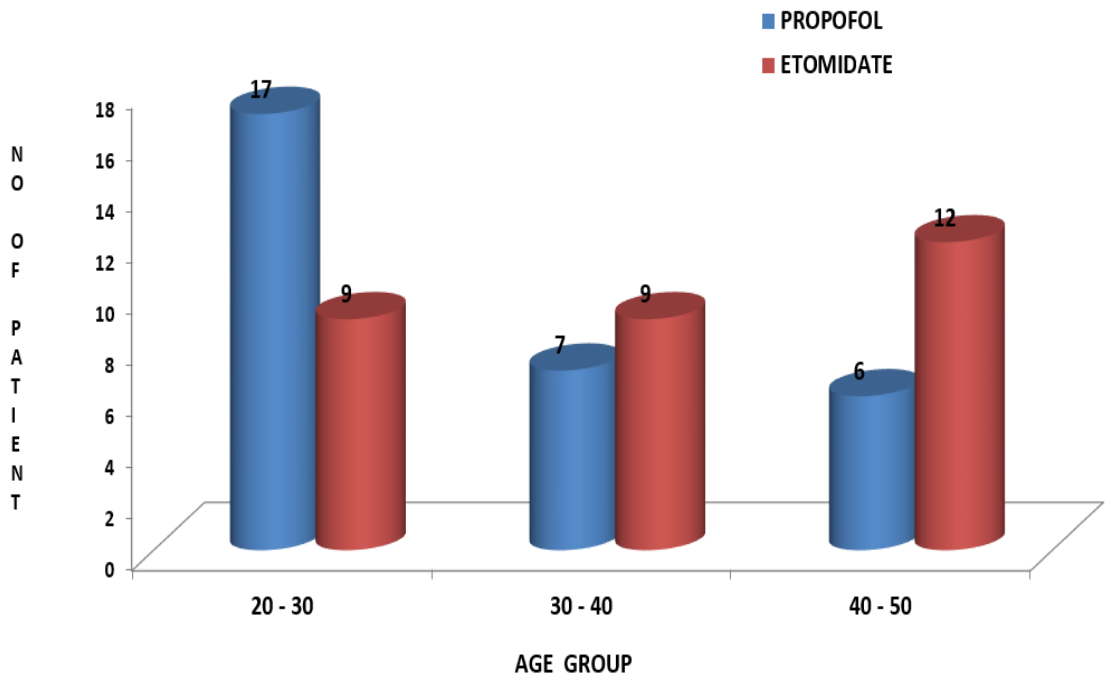


TABLE- 2

AGE DISTRIBUTION

Age	PROPOFOL		ETOMIDATE	
	NUMBER	%	NUMBER	%
20 – 30	17	56.67	9	30
30 – 40	7	23.33	9	30
40 – 50	6	20	12	40
TOTAL	30	100	30	100
Mean ± SD	32.03 ± 14.07		33.07± 10.01	
t-value	0.33			
Df	58			
p-value	0.074* (Not Significant)			

*P value >0.05 Not Significant

Patient in the age group of 20 to 50 years were included in the study. Propofol group had a mean age of 32.03 ±14.07 years and Etomidate group with the mean age of 33.07± 10.01 years. The mean age was comparable in both the groups with a P value of 0.074.

CHART-3

WEIGHT

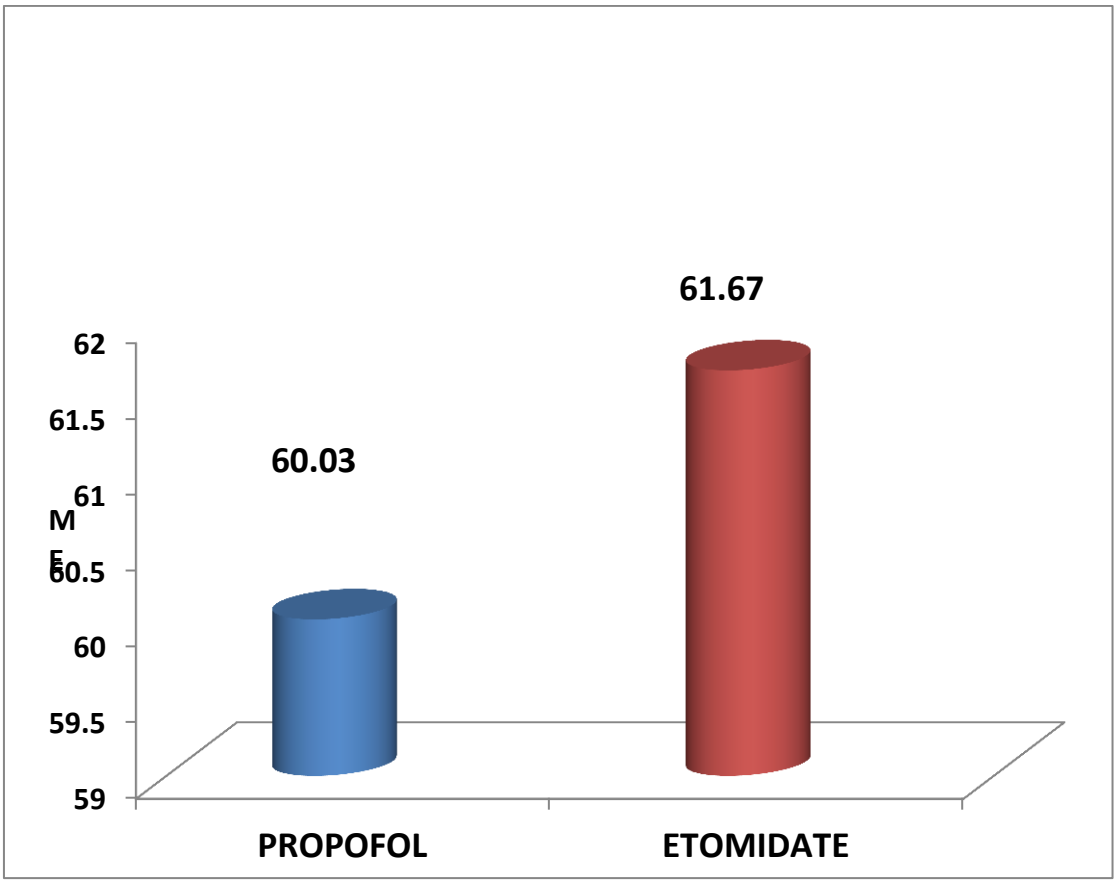


TABLE-3

WEIGHT

PROPOFOL		ETOMIDATE		t-value	Df	p-value
MEAN	SD	MEAN	SD			
60.03	6.52	61.67	6.98	0.94	58	0.06*

*P value >0.05 Not Significant

With respect to weight both the groups were comparable with each other.

Mean weight of propofol group is 60.03±6.25 and etomidate 61.67± 6.98 the p value is 0.06 which is statistically not significant.

CHART-4

ASA GRADE

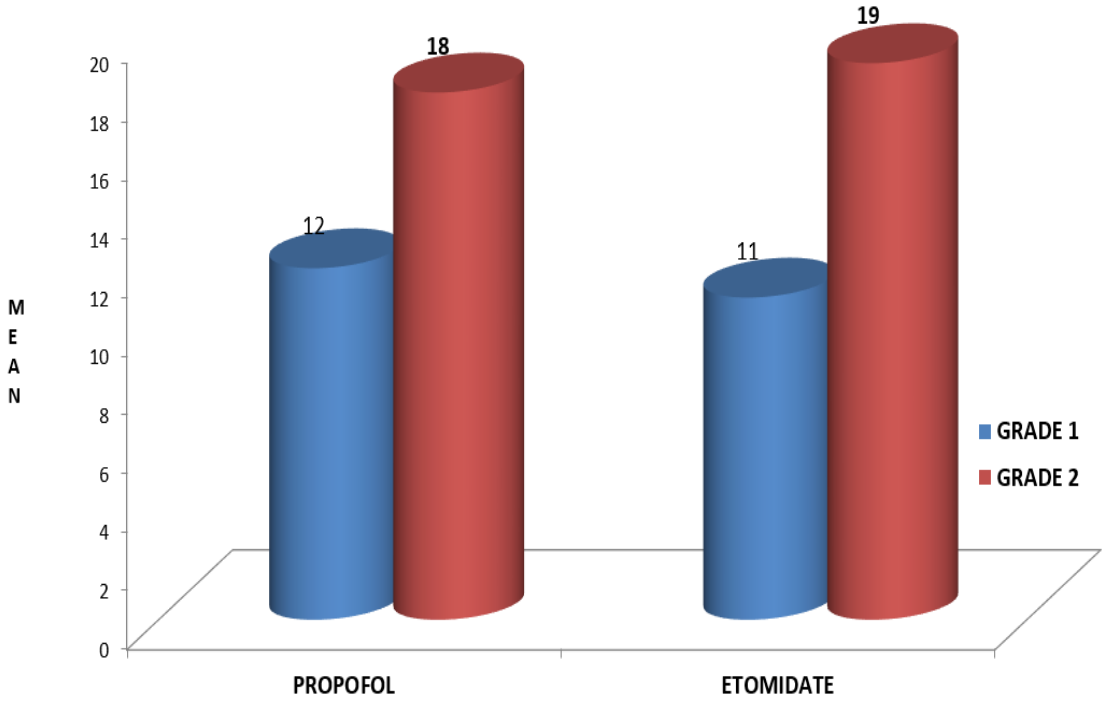


TABLE-4

ASA GRADE

ASA GRADE	PROPOFOL		ETOMIDATE	
	NO.	%	NO.	%
1	12	70.00	11	36.67
2	18	30.00	19	63.33
TOTAL	30	100	30	100
Chi square value	0.70			
Df	58			
p-value	**0.79* (Not Significant)**			

*P value >0.05 Not Significant

American society of Anaesthesiologist physical status were comparable in both the groups with the p value of 0.79. Thus statistically not significant.

CHART 5

HEART RATE

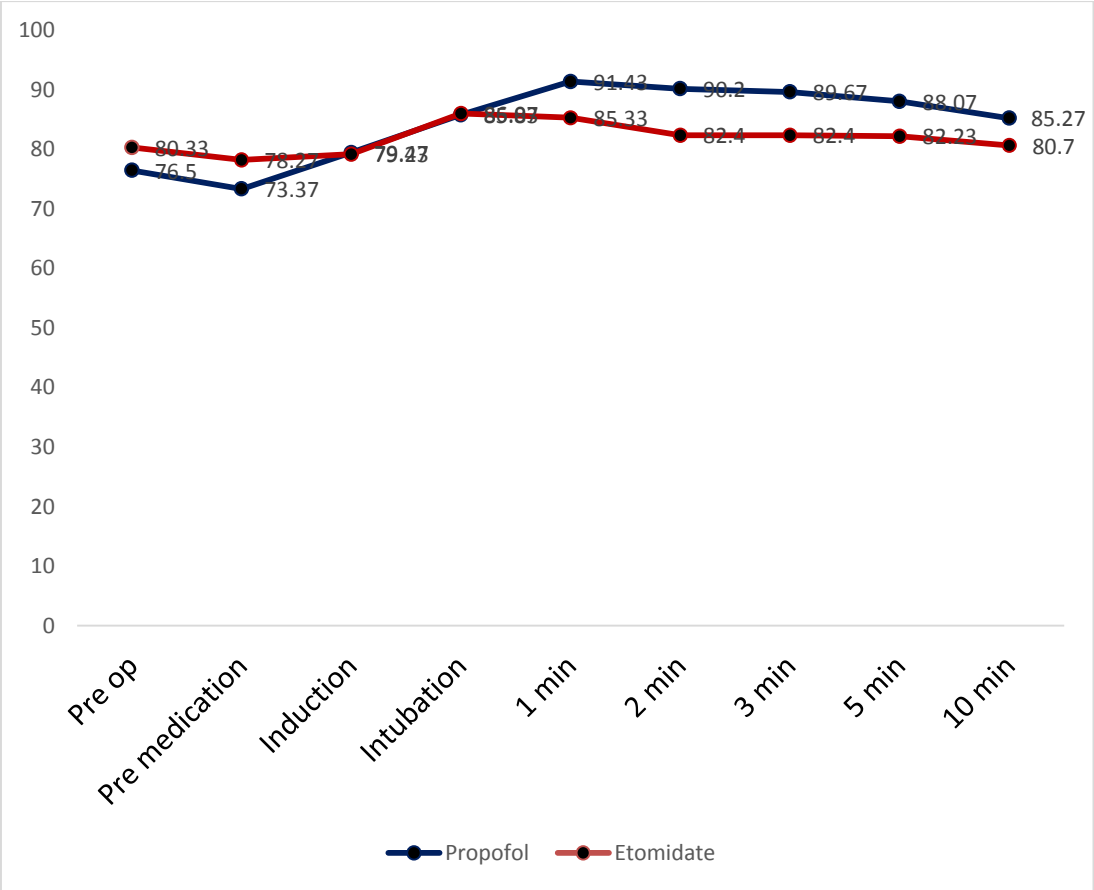


TABLE-5

HEART RATE

Variables	PROPOFOL		ETOMIDATE		t-value	Df	p-value
	MEAN HR	SD	MEAN	SD			
PRE OP	76.50	10.04	80.33	9.42	1.53	58	0.13*
PRE MEDICATION	73.37	9.35	78.27	9.29	2.04	58	0.05
INDUCTION	79.47	11.62	79.23	9.64	0.09	58	0.93*
INTUBATION	85.83	20.14	86.07	8.71	0.06	58	0.95*
1 MIN	91.43	13.17	85.33	7.93	2.17	58	0.03
2 MIN	90.20	12.73	82.40	9.48	2.69	58	0.01
3 MIN	89.67	9.46	82.40	9.92	2.91	58	0.01
5 MIN	88.07	9.47	82.23	10.40	2.27	58	0.03
10 MIN	85.27	11.84	80.70	8.95	1.69	58	0.10*

. p value < 0.05: Significant * p value > 0.05 : Not Significant

Heart rate in both propofol and etomidate group increased after intubation compared to the values at induction. In the propofol group, the heart rate increased by seven to twelve beats per minute. In the etomidate group the heart rate increased by three to seven beats per minute, which was measured during intubation and 1, 2, 3, and 5 mins after intubation. The difference was statistically significant.

CHART-6

SYSTOLIC BLOOD PRESSURE

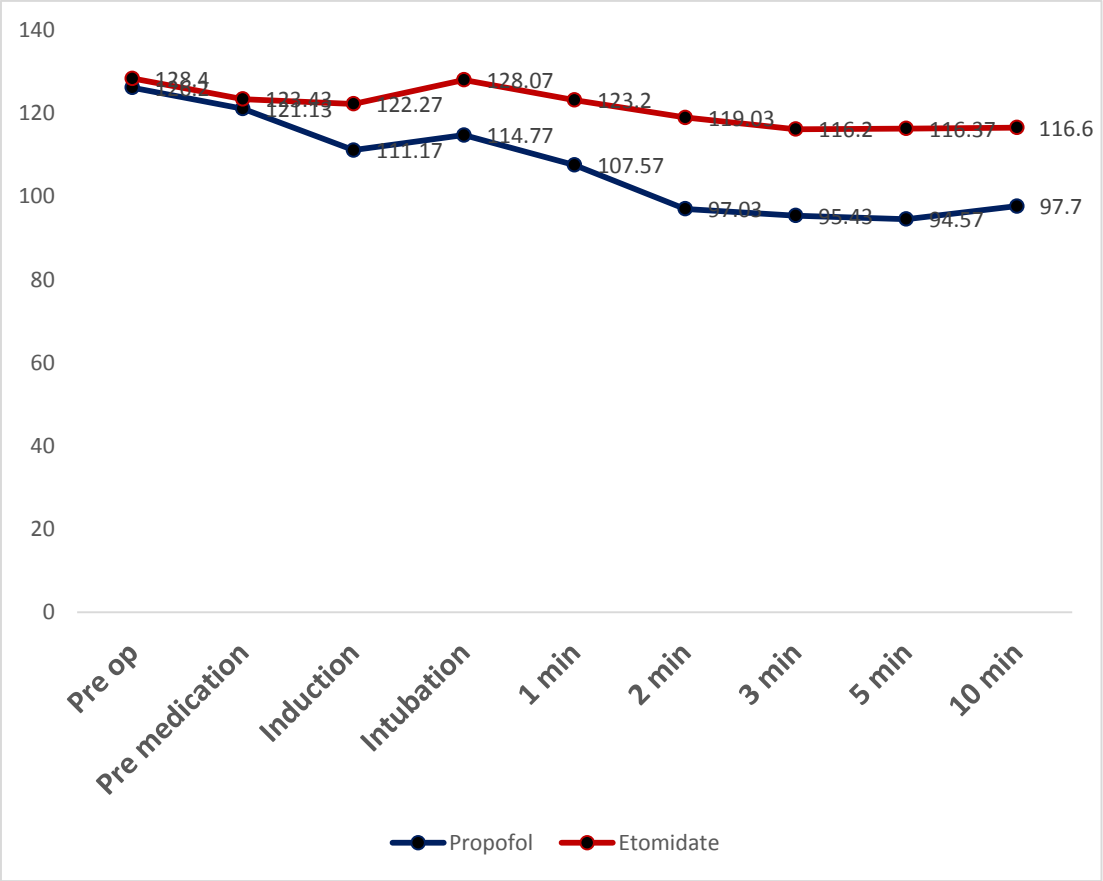


TABLE-6

SYSTOLIC BLOOD PRESSURE

Variables	PROPOFOL		ETOMIDATE		t-value	Df	p-value
	MEAN SBP	SD	MEAN SBP	SD			
PRE OP	126.20	9.07	128.40	7.67	1.01	58	0.32*
PRE MEDICATION	121.13	9.93	123.43	7.31	1.02	58	0.31*
INDUCTION	111.17	9.05	122.27	6.36	5.50	58	0.001
INTUBUTION	114.77	12.09	128.07	7.75	5.07	58	0.001
1 MIN	107.57	18.03	123.20	7.36	4.40	58	0.001
2 MIN	97.03	8.34	119.03	8.60	10.04	58	0.001
3 MIN	95.43	6.71	116.20	6.66	12.03	58	0.001
5 MIN	94.57	5.35	116.37	7.37	13.10	58	0.001
10 MIN	97.70	7.95	116.60	7.18	9.66	58	0.001

. p value < 0.05 : Significant * p value > 0.05 : Not Significant

When compared with systolic blood pressure values at the induction, there was a greater change in the propofol group at intubation as well as 1, 2, 3, 5 and 10 mins after intubation. The difference was statistically significant with respect to the etomidate group during the same period with a p value of < 0.05.

CHART 7

DIASTOLIC BLOOD PRESSURE

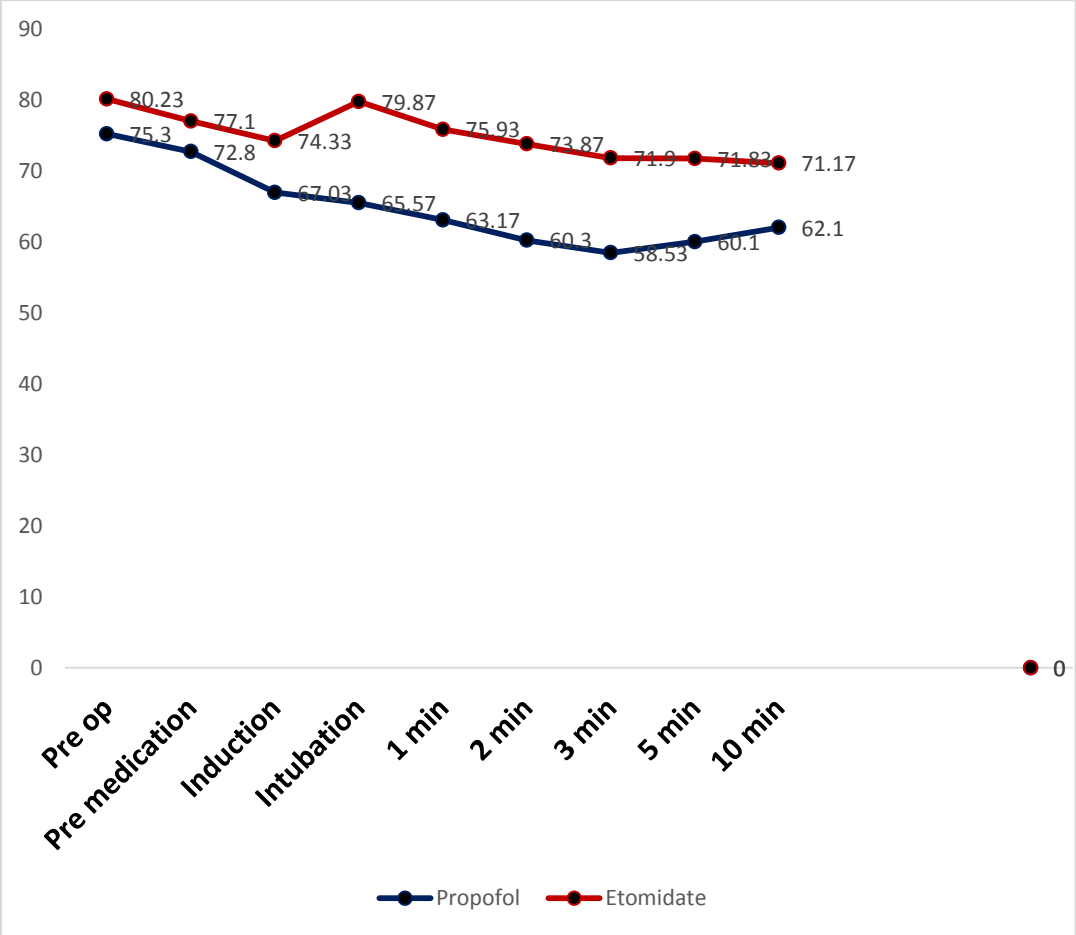


TABLE-7

DIASTOLIC BLOOD PRESSURE

Variables	PROPOFOL		ETOMIDATE		t-value	Df	p-value
	MEAN DBP	SD	MEAN DBP	SD			
PRE OP	75.30	10.38	80.23	10.27	1.85	58	0.07*
PRE MEDICATION	72.80	9.83	77.10	8.86	1.78	58	0.08*
INDUCTION	67.03	7.65	74.33	8.52	3.46	58	0.001
INTUBATION	65.57	15.27	79.87	10.87	4.18	58	0.001
1 MIN	63.17	7.00	75.93	9.66	5.86	58	0.001
2 MIN	60.30	5.60	73.87	8.46	7.32	58	0.001
3 MIN	58.53	5.26	71.90	7.10	8.28	58	0.001
5 MIN	60.10	6.31	71.83	6.37	7.17	58	0.001
10 MIN	62.10	4.91	71.17	5.94	6.45	58	0.001

. p value < 0.05: Significant * p value > 0.0 5: Not Significant.

Following intubation the change in diastolic blood pressure was more in propofol group compared to etomidate group with respect to the values at induction. The difference between the two groups was statistically significant with the p value of < 0.05.

CHART-8

MEAN ARTERIAL BLOOD PRESSURE

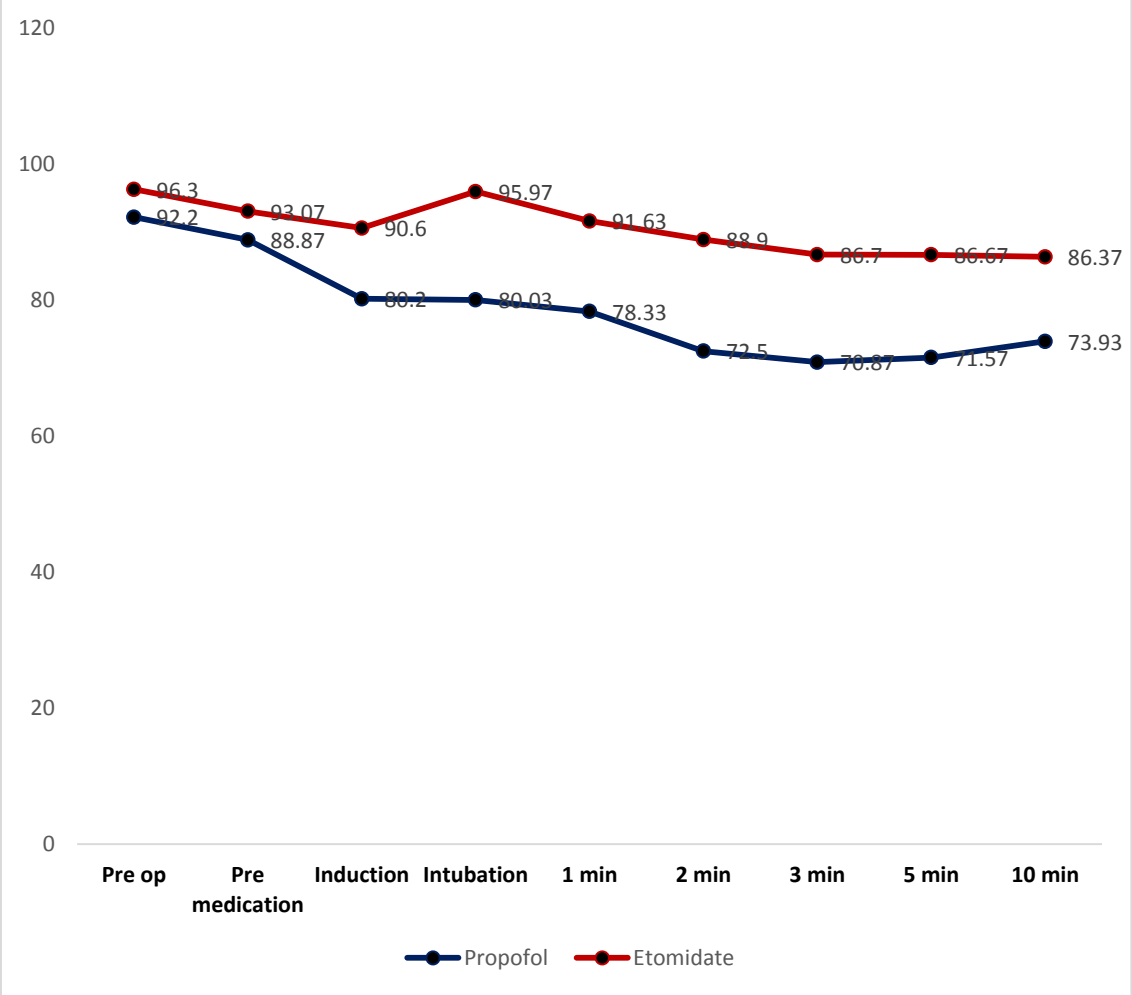


TABLE-8

MEAN ARTERIAL BLOOD PRESSURE

Variables	PROPOFOL		ETOMIDATE		t-value	Df	p-value
	MEAN MAP	SD	MEAN MAP	SD			
PRE OP	92.20	9.41	96.30	8.60	1.76	58	0.08*
PRE MEDICATION	88.87	9.02	93.07	7.30	1.98	58	0.05
INDUCTION	80.20	11.28	90.60	7.21	4.26	58	0.001
INTUBUTION	82.03	12.01	95.97	8.74	5.14	58	0.001
1 MIN	78.33	8.10	91.63	7.94	6.42	58	0.001
2 MIN	72.50	5.31	88.90	7.09	10.14	58	0.001
3 MIN	70.87	4.81	86.70	5.37	12.04	58	0.001
5 MIN	71.57	5.39	86.67	6.09	10.18	58	0.001
10 MIN	73.93	4.76	86.37	5.14	9.72	58	0.001

. p value < 0.05 : Significant * p value > 0.05 : Not Significant

Mean arterial pressure is diastolic blood pressure + 1/3 of systolic blood pressure. The trend in Mean Arterial Pressure was similar to the trend in diastolic blood pressure. After intubation and 1, 2, 3, 5 and 10 mins afterwards the mean arterial pressure values were compared in etomidate as well as propofol groups. The mean arterial pressure decreased by maximum of 9 mm of Hg in propofol group, whereas the mean arterial pressure deceased by maximum of 4 mm of Hg in etomidate group. The difference between the two groups was statistically significant with p value of < 0.05.

Discussion

DISCUSSION

Rapid induction and hemodynamic stability with minimal side effects are the most important characteristics desired from an ideal induction agent.

In this study we compared the hemodynamic response to endotracheal intubation using etomidate and propofol as induction agents in 60 patients with 30 patients in each group, within the age group of 20-50 years of either sex, weighing 40-80 Kg.

Regarding the underlying variables such as gender, age, weight and ASA physical status of the patients, there was no significant difference, thus the compounding effects of these variables had been neutralized. Regarding the weight, the propofol group had a mean of 60.03, and etomidate group had a mean of 61.03 but this difference between the groups, was not statistically significant.

The baseline and premedication values of systolic blood pressure, diastolic blood pressure, mean arterial pressure, were comparable in both the groups (table **6, table 7, table 8)**. Following intubation with propofol, there were significant changes in systolic blood pressure, diastolic blood pressure and mean arterial pressure compared to etomidate group and p

values at various time intervals remained significant (<0.05). This hypotension with propofol due to decrease in preload, was managed with fluids, and by decreasing concentration of inhalation agent.

Hug et al[33] conducted a study in 25,000 patients, he found out that propofol caused bradycardia in 4.2% patients and hypotension in 15.7% patients. In our study there was no incidence of bradycardia but significant hypotension occurred in 6 patients out of 30 patients which is around 20% which is comparable with the above study.

The effect of etomidate and propofol on heart rate is controversial. According to studies of Siedy J et al[27] , Ghafor et al[28] and Kaur et al[29] , Mean heart rate was comparable in both the groups. Heart reate may increase or decrease, or these changes can be minimal following induction with these agents. The reason for this difference is not clear.

In the studies of Ulsamer et al[30], Moffat et al[31] they found that Etomidate was associated with unacceptably sudden increase in heart rate, while Shah et al[9] reported sustained increase in heart rate with Propofol. In our study, the change in heart rate was not much significant at induction and intubation in both the groups but etomidate was found to maintain heart rate within range of 3-7 beats per minute at 1,2,3,5 mins following intubation **(table:5).**

In the study of Kahlon A et al[12], they found that etomidate caused myoclonus around 76% in placebo group, 44% in lignocaine group and 28% in midazolam group . In our study myoclonus was observed in 4 out of 30 patients (13.33%) who were induced with etomidate, while no equivalent signs were noted in propofol group. This finding correlates with the above study.

Picard P et al[32] did a study on 6264 patients which showed that on an average, 70% of patients complained, pain on injection. In our study, 22 patients out of 30 patients complained of pain on injection(73.33%).

Shah et al[9], Masoudifar[13] and Beheshtian, Aggarwal[7] et al, MeenaKumari[6], all the above studies showed that the changes in systolic blood pressure, diastolic blood pressure and mean arterial pressure were less in etomidate group compared to propofol group which is in total agreement with our study **(table 6,7 &8).**

There was no incidence of nausea and vomiting in both the groups. No other complications were noted in both etomidate and propofol group.

Limitation of the Study

LIMITATION OF **STUDY**

The study design had some limitation. We did not measure plasma cortisol and adrenocorticotrophic hormone level due to nonavailability of the above tests in our institution.

Summary

SUMMARY

The aim of the study is to compare etomidate and propofol induction on hemodynamic response to laryngoscopy and intubation. The study was conducted in 60 ASA I & II patients in the age group of 20 to 50 years who were posted for elective cases under general anaesthesia. Their baseline heart rate, systolic blood pressure, diastolic blood pressure, SpO2 and ETCO2 were noted. Both the groups were premedicated with iv glycopyrrolate 0.05mcg/kg, iv midazolam 0.025mg/Kg, iv fentanyl 2mcg/Kg. Following premedication the above variables were again noted. Group A patients were induced with iv propofol at the dose of 2.5mg/Kg and Group B patients received iv etomidate at 0.3mg/Kg. In both the groups, Injection vecuronium was given at the dose 0.08mg/Kg and they were maintained with O2:N2O =33%:66% and Isoflurane 1% dial concentration. Laryngoscopy was performed by trained anesthesiologists after 5 mins. Duration of laryngoscopy was kept at a maximum of 10 seconds. Trachea was intubated with appropriate size endotracheal tube. The variables (HR, SBP, DBP, MAP, SpO2) were measured during induction, intubation and post intubation at intervals of 1,2,3,5 and 10 mins.

On comparing the two groups, the following results were obtained –

1) Age, Sex, Weight and ASA status were comparable in both the groups.

2) Propofol was found to produce hypotension in more or less 20%-30% of patients irrespective of the underlying condition.

3) Etomidate was found to maintain hemodynamic stability though there were no significant difference in heart rate variability in both the groups during laryngoscopy and intubation.

4) Myoclonus was seen in 4 out of 30 patients induced with Etomidate, pain on injection was more common with Propofol.

Conclusion

CONCLUSION

As per the results of the study, Propofol produced more hemodynamic changes than Etomidate. Thus we conclude that Etomidate is more stable in terms of hemodynamic stability.

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Annexures

PATIENT CONSENT FORM

Study title

'Comparison of Propofol and Etomidate induction on haemo dynamic response to endotracheal intubation'

Study centre

ESIC Medical College & PGIMSR, K. K. Nagar, Chennai -78

Participant's Name:

Age: Sex:

Diagnosis:

Plan:

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the pitfall in the procedure. I have been explained about the safety, advantage and disadvantage of the technique. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

I have been explained that the anaesthetic technique is a standard and approved technique. This may help in future research in the field of anaesthesia. I consent to undergo this procedure.

Insurance No: Signature / thumb impression of patient

Date :

ஒப்புதல் படிவம்

1. எனக்கு அறுவை சிகிச்சையை செய்யுமாறு மருத்துவர் மற்றும் குழுவினரை வேண்டிக் கொள்கிறேன்.

2. நோயின் தன்மை :

   சிகிச்சை முறை :

   இவை அனைத்தும் எனக்கு மருத்துவர் □ லம் தெளிவாக விளக்கப் பட்டன.

3. எனக்கு முழு மயக்கத்தின்போது புரோப்போபல் மற்றும் இடோமிடேட் (Propofol & Etomidate) என்ற மயக்க மருந்து கொடுத்து அறுவை சிகிச்சை மற்றும் ஆய்வு செய்துகொள்ள சம்மதிக்கிறேன்.

4. இவற்றின் பின் விளைவுகளை மருத்துவர் □ லம் அறிந்து கொண்டேன்.

5. அனைத்து மருத்துவ சிகிச்சை முறைகளின் நிறைகளும் குறைகளும் எனக்கு விளக்கப்பட்டன.

6. மேலே கொடுக்கப்பட்டுள்ள அனைத்தும் மருத்துவமனை நன்னெறி (Ethics) குழுவின் வரைமுறைக்கு உட்பட்டே நடக்கும் என மருத்துவர் விளக்கினார். மேலும் இந்த சிகிச்சை முறைகளுக்கு உடன்பட மறுக்கவும் எனக்கு உரிமை உண்டு என்பதை அறிவேன்.

7. என் சிகிச்சையின்போது கிடைக்கும் தகவல்களை மருத்துவ ஆராய்ச்சிக்கு பயன்படுத்தவும் அளிக்கிறேன்.

நான் இந்த ஒப்புதல் படிவத்தை படித்த பின்னரே / படித்து காண்பிக்கப்பட்ட பின்னரே, இதன் சாராம்சத்தை முழுவதுமாக புரிந்துகொண்ட பின்னே முழுமனதுடன் சம்மதித்து கையெழுத்திடுகின்றேன்.

சாட்சி **ஒப்புதல் அளிப்பவர்**

PROFORMA

Name of the patient:

Age:

Sex:

Weight:

Height:

Insurance No:

Group:

Date:

Procedure:

Anaesthetic plan:

PRE OPERATIVE DETAILS
ASA GRADING
MALLAMPATTI GRADING
REMARKS

PRE OPERATIVE				
HR:	**SBP:**	**DBP:**	**MAP:**	**SPO2**

INTRAOPERATIVE

DRUGS:

Vitals	Pre Op	Pre Med	Induction	Intubation	1 Min	2 Min	3 Min	5 Min	10 Min
HR									
SBP									
DBP									
MAP									

MASTER CHART

SL NO	GROUP	NAME	AGE	SEX	WEIGHT	DIAGNOSIS	SURGERY	ASA	HEART RATE (/min)								SYSTOLIC BLOOD PRESSURE (mmHg)								DIASTOLIC BLOOD PRESSURE (mmHg)								MEAN ARTERIAL PRESSURE (mmHg)								SPO2							
									PREOP	PREMEDICATION	INDUCTION	INTUBUTION	1	2	3	5	10	PREOP	PREMED	INDUCTION	INTUBUTION	1	2	3	5	10	PREOP	PREMED	INDUCTION	INTUBUTION	1	2	3	5	10	PRE OP	PREMED	INDUCTION	INTUBUTION	1	2	3	5	10	PREOP	PREMED	INDUCTION	INTUBUTION	1	2	3	5	10
1	E	MALATHY	35	F	62	ACUTE CHOLECYSTITIS	LAP CHOLECYSTECTOMY	2	77	80	83	89	89	88	84	92	88	130	125	120	121	124	118	114	127	124	101	87	82	86	87	84	84	85	80	111	100	95	98	99	95	94	99	95	100	100	99	100	99	100	98	100
2	P	MANIKANDAN	29	M	78	RT GYNECOMASTIA	WEBSTER'S PROCEDURE	2	62	58	83	102	90	94	95	92	90	138	111	132	117	109	105	101	100	97	96	72	81	78	81	71	66	65	64	110	85	98	91	90	82	78	77	75	98	99	100	98	100	99	98	100	99
3	E	JAYAPAUL	40	M	78	RT RENAL CALCULI	RT PCNL	2	84	81	83	91	81	74	73	66	68	136	134	132	131	144	111	116	107	102	98	96	99	94	91	81	81	68	67	111	109	110	106	109	91	93	81	79	98	100	100	98	100	100	98	99	100
4	P	SAJEDHA	32	F	60	CAUDA EQUINA SYNDROME	DISCECTOMY	2	93	92	111	116	127	133	104	99	116	124	115	105	108	106	85	96	107	115	84	90	73	85	66	65	65	77	77	97	98	84	93	79	72	75	87	90	98	100	98	98	98	100	98	100	100
5	E	KRIHNA MOORTHY	36	M	57	LT RENAL CALCULUS	LT PCNL	1	56	60	58	70	73	52	56	55	60	130	117	116	130	120	112	108	105	109	90	91	85	93	91	92	87	77	80	103	100	95	105	101	99	94	86	90	99	99	98	99	98	99	100	99
6	E	SATISH	25	M	55	RT RENAL CALCULI	RT PCNL	1	93	92	96	102	98	91	87	77	74	122	120	116	126	127	118	119	113	108	90	85	69	99	96	75	75	72	67	101	97	85	108	106	89	90	86	81	99	99	98	99	98	98	99	99	99
7	P	ASHWINI	26	F	54	HEMANGIOMA SCALP	EXCISION	1	68	76	90	80	105	103	106	95	80	140	142	121	104	102	86	84	96	95	90	93	68	51	50	61	51	65	63	107	109	86	69	67	69	62	75	74	100	98	99	100	99	98	100	99	98
8	E	THIYAGARAJAN	50	M	60	RT CSOM	RT CORTICAL MASTOIDECTOMY	2	84	88	86	96	88	89	93	92	89	123	108	118	116	113	110	106	104	110	74	79	73	81	71	66	65	64	68	90	89	88	93	85	81	79	77	82	100	98	99	100	99	99	100	98	98
9	P	SOMAIAH	22	M	60	RT CSOM	ST CORTICAL MASTOIDECTOMY	1	82	84	79	118	118	75	71	76	72	130	133	103	107	102	107	102	100	95	88	88	59	65	62	65	62	57	57	102	103	74	79	75	79	75	71	70	99	98	100	99	100	99	99	98	98
10	E	SHANMUGHAM	33	M	65	FUNGAL SINUSITIS	FESS	2	68	72	69	81	71	69	63	90	83	130	123	123	153	136	144	115	114	100	90	83	84	104	90	90	83	81	72	103	96	97	120	105	108	94	92	81	99	99	100	99	100	100	99	98	99
11	P	SUGANYA	30	F	63	PRIMARY INFERTILITY	DIAGNOSTIC LAPROSCOPY	1	71	68	69	88	86	84	81	81	80	134	128	100	98	88	92	96	97	110	90	83	80	78	68	71	68	72	70	105	98	87	85	75	78	77	80	83	98	99	99	98	99	100	98	99	99
12	E	SHANKARI	47	F	62	MULTI NODULAR GOITRE	TOTAL THYROIDECTOMY	2	81	78	78	88	83	81	80	78	76	146	138	134	140	130	127	119	122	118	88	81	80	98	81	84	78	77	71	107	100	98	112	97	98	92	92	87	98	100	99	98	99	99	98	99	100
13	E	ILAKKIYAMATHI	26	F	65	#CLAVICLE	ORIF	1	75	78	80	86	87	85	80	87	83	146	130	121	124	118	115	110	125	120	98	90	78	80	81	80	80	81	77	114	103	92	95	93	92	90	96	91	98	98	98	98	98	99	98	100	98
14	E	NEELAVATHI	28	F	57	RT THYROID NODULE	RT HEMI THYROIDECTOMY	2	88	87	80	92	82	82	86	88	82	132	118	128	126	123	120	116	114	120	74	79	73	81	71	66	65	64	68	93	92	91	96	88	84	82	81	85	99	100	98	99	98	98	99	98	100
15	P	DAIVANAYAKI	38	F	55	PRIMARY INFERTILITY	DIAGNOSTIC LAPROSCOPY	2	64	61	85	100	92	96	92	91	94	128	101	122	107	99	95	90	90	91	85	72	71	70	71	61	60	58	58	99	82	88	82	80	72	70	69	69	99	98	98	99	98	98	99	100	98
16	P	ILAVARASI	45	F	59	L4-L5 DISC PROLAPSE	L4-L5 DISCECTOMY	2	66	72	88	78	98	90	92	85	72	134	136	117	98	96	84	86	92	90	85	90	65	57	50	56	54	62	65	101	105	82	71	65	65	65	72	73	100	98	99	100	99	98	100	98	98
17	E	SATHAYANARAYANAN	49	M	72	#CLAVIVLE LT	ORIF	2	90	88	96	98	88	80	87	92	91	123	119	127	124	120	117	115	119	120	90	84	80	81	87	80	77	75	74	101	96	96	95	98	92	90	90	89	98	98	99	98	98	98	98	98	98
18	P	KANHU CHARAN	42	M	65	RT PARASYMPHYSIS#	ORIF	1	68	64	66	70	68	73	77	75	78	123	120	121	109	104	97	99	100	103	78	72	70	68	60	58	58	60	62	93	88	87	82	75	71	72	73	76	99	99	100	99	100	99	99	98	99
19	E	SOFIA EZHILARASI	42	F	65	RT LOBE SOLITARY NODULE THYROID	RT HEMI THYROIDECTOMY	2	71	73	72	77	81	76	78	75	74	123	118	126	121	128	124	121	124	126	80	78	77	81	76	74	72	71	70	94	91	93	94	93	91	88	89	89	100	99	98	100	98	100	100	99	99
20	P	NAVANEETH KUMAR	28	M	70	RT GYNECOMASTIA	RT WEBSTER'S PROCEDURE	2	59	61	59	70	77	76	70	68	66	114	116	106	90	88	84	98	99	107	68	66	64	60	58	55	57	60	61	83	83	78	70	68	65	71	73	76	100	100	99	100	99	98	100	99	100
21	P	ARUL PRAVEEN	26	M	52	R LT INFERIOR TURBINATE HYPERTROF	SEPTOPLASTY	2	72	70	77	78	92	94	93	91	88	127	125	119	120	107	100	96	98	101	64	66	62	60	58	55	51	50	57	85	86	81	80	74	70	66	66	72	98	100	100	98	100	99	98	100	100
22	E	RAJESH	27	M	72	RT MAXILLARY POLYP ,DSR	FESS WITH SEPTOPLASTY	2	80	82	87	88	90	91	88	86	84	134	128	127	135	127	125	120	121	126	77	75	71	77	70	68	70	72	71	96	93	90	96	89	87	87	88	89	98	99	100	98	100	100	98	100	99
23	P	VAISHNAVI	26	F	57	CHRONIC TONSILLITIS	TONSILLECTOMY	1	71	70	65	73	74	77	82	88	80	119	110	109	117	105	100	96	90	101	64	64	60	58	55	52	55	56	60	82	79	76	78	72	68	69	67	74	99	99	98	99	98	100	99	99	99
24	E	SARATH	46	M	64	LT PUJ OBSTRUCTION	LT PYELOPLASTY	1	88	89	85	92	91	88	86	84	81	140	136	134	144	137	139	132	128	130	90	88	85	90	82	78	77	80	84	107	104	101	108	100	98	95	96	99	99	98	100	99	100	98	99	99	98
25	P	GEETHA	37	F	57	YSPLASIA OF RT LAT BORDER OF TONGU	WLE	1	76	73	77	75	81	83	87	83	80	119	116	100	109	107	90	88	92	98	67	67	68	70	70	68	63	58	59	84	83	79	83	82	75	71	69	72	100	98	100	100	100	100	100	98	98
26	P	KANAN	20	M	59	RT SUBMANDIBULAR SIALONIDITIS	SUBMANDIBULAR SIALADENACTOMY	2	93	91	90	104	99	100	101	99	96	120	124	111	128	103	100	94	90	98	71	70	68	72	66	60	55	58	57	87	88	82	91	78	73	68	69	71	98	98	99	98	99	100	98	98	98
27	P	LOGESH	22	M	62	DNS	SEPTOPLASTY	2	68	64	71	79	81	84	88	82	78	126	120	107	134	115	100	98	90	95	72	74	68	70	66	55	54	53	55	90	89	81	91	82	70	69	65	68	99	99	99	99	99	99	98	99	
28	P	ASHA	27	F	50	DERMOID CYST FOREHEAD	EXCISION	2	71	70	63	90	84	83	78	77	75	140	134	111	144	119	104	100	98	96	82	80	71	84	71	60	63	67	69	101	98	84	104	87	75	75	77	78	100	99	98	100	99	99	100	99	99
29	P	PRAKASH	29	M	53	DSL	SEPTOPLASTY	1	83	80	79	101	98	90	93	96	91	134	130	123	137	119	104	100	102	110	74	70	68	77	60	58	55	60	64	94	90	86	97	80	73	70	74	79	100	100	98	100	100	98	100	99	100
30	E	LATHAMARY	36	F	54	IMPACTED TOOTH	SURGICAL EXTRACTION	2	84	86	80	90	92	91	93	91	88	128	124	120	133	130	128	120	124	121	75	71	70	77	72	71	68	69	67	93	89	87	96	91	90	85	87	85	98	98	98	98	98	98	100	98	
31	P	SOKKAMMAL	24	M	58	LT EAR ATRESIA	LT REVISION MEATOPLASTY	1	78	77	74	4	70	68	77	68	60	118	116	90	107	92	84	80	88	92	72	71	60	71	61	55	58	56	60	87	86	70	83	71	65	65	67	71	98	99	99	98	99	98	98	98	99
32	E	VEERARAGAVAN	33	M	60	LT CSOM	LT CORTICAL MASTOIDECTOMY	2	78	78	76	84	80	82	83	81	79	128	123	120	134	131	128	122	119	120	81	74	80	76	77	74	71	78	70	97	90	93	95	95	92	88	92	87	98	100	99	98	100	99	98	99	100
33	E	JOTHIRAMAN	37	M	67	RT #CLAVICLE	ORIF	2	71	73	71	79	83	79	80	78	77	123	120	118	128	117	124	120	119	119	71	70	68	74	70	68	68	70	71	88	87	85	92	86	87	85	86	87	99	100	100	99	100	99	99	100	100
34	P	BANUPRIYA	24	F	63	SNT LT LOBE	TOTAL THYROIDECTOMY	1	76	77	70	89	95	101	99	87	84	119	116	104	110	103	97	94	90	88	68	65	60	67	58	55	54	50	57	85	82	75	81	73	69	67	63	67	99	98	98	99	98	100	99	100	98
35	P	RAFI	25	M	62		B/L WEBSTERS	2	68	64	62	70	76	82	87	89	74	117	112	100	114	191	90	88	86	90	66	66	60	64	58	55	58	60	62	83	81	73	81	102	67	68	69	71	100	99	100	100	99	99	100	98	99
36	E	RAJESHWARI	35	F	74	PLANNED PARENTHOOD	LAP STERILIZATION	2	63	60	63	71	77	70	68	68	67	128	125	124	129	120	119	118	117	120	71	70	68	70	68	66	66	68	70	90	88	87	90	85	84	83	84	87	98	98	98	98	98	98	100	98	
37	P	VELAMMAL	36	F	76	CHRONIC TONSILLITIS	TONSILLECTOMY	2	94	86	85	93	104	101	99	95	99	104	102	100	117	109	104	98	90	96	60	62	60	58	60	58	55	50	60	75	75	73	78	76	73	69	63	72	99	99	99	99	99	98	99	98	
38	E	VINOD	27	M	56	LEFT COLLOID GOITRE	LEFT HEMITHYROIDECTOMY	1	82	74	74	82	83	85	80	77	74	117	115	114	121	118	109	106	110	116	77	72	69	77	74	77	71	70	68	90	86	84	92	89	88	83	83	84	99	100	99	99	100	99	99	99	100
39	E	LALITHA	33	F	60	DENTAL CYST	CYST EXCISION	2	69	60	64	70	73	70	71	68	70	132	130	125	127	120	121	124	119	120	68	64	60	65	60	68	60	62	60	89	86	82	86	80	86	81	81	80	100	100	100	100	100	99	100	100	100
40	E	MANIVANNAN	35	M	64	LT TORN EAR	LT EAR RECONSTRUCTION	2	78	74	71	79	82	80	79	78	77	117	114	110	120	117	110	108	114	115	60	58	55	64	60	62	65	66	60	79	77	73	83	79	78	79	82	78	100	98	98	100	98	100	100	100	98
41	P	SARALA	36	F	60	GRADE3 DIFFUSE GOITRE	TOTAL THYROIDECTOMY	2	75	70	73	88	89	79	86	77	75	119	115	110	102	98	90	86	89	92	60	55	57	64	60	58	57	60	61	80	75	75	77	73	69	67	70	71	99	98	99	99	98	98	99	98	98
42	E	GEETHARANI	29	F	58	LT CSOM	LT CARTILAGE TYMPANOPLASTY	1	92	87	93	99	104	101	104	103	102	132	128	120	122	117	109	103	100	114	81	78	69	65	64	60	62	60	64	98	95	86	84	82	76	76	73	81	99	98	100	99	98	99	99	98	98
43	E	HEMANTH KUMAR	21	M	60	AC POLYP	FESS	1	78	76	76	83	80	82	85	81	78	127	123	119	129	119	114	117	110	108	77	72	70	81	77	74	70	68	70	94	89	86	97	91	87	86	82	83	98	99	100	98	99	100	98	98	99
44	P	SHOBANA	32	F	71	DENTAL CYST	PERIAPICAL SURGERY	2	96	90	98	105	101	103	99	107	100	140	134	115	123	119	110	104	100	116	80	83	74	80	70	68	66	60	61	100	100	88	94	86	82	79	73	79	98	99	98	98	99	100	98	99	99
45	E	LAKSHAMANAN	46	M	80	LIPOMA B/L TEMPOROCCIPITAL REGION	LIPOMA EXCISION	1	80	74	74	81	82	86	83	80	78	129	126	118	120	114	112	117	119	115	75	72	68	73	70	68	66	69	71	93	90	85	89	85	83	83	86	86	98	100	98	98	100	98	98	99	100
46	P	SRINIVASAN	28	M	67	CHRONIC DACRYOCYSTITIS	ENDO DCR	1	89	82	94	99	103	100	99	101	104	125	121	114	117	103	104	98	96	107	88	80	71	77	70	68	60	62	58	100	94	85	90	81	80	73	73	74	99	100	98	99	100	98	99	100	100
47	E	NELAVENI	27	F	58	CHRONIC SINUSITIS	FESS	1	95	92	90	96	99	90	91	93	94	118	116	110	120	116	108	105	109	111	80	75	71	82	77	74	70	71	70	93	89	84	95	90	85	82	84	84	99	99	99	99	99	98	99	100	99
48	E	KOLLAPURI	41	M	56	RIGHT COLLOID GOITRE	RIGHT HEMITHYROIDECTOMY	1	88	80	87	91	90	93	89	91	86	132	133	128	127	121	119	123	117	120	88	84	80	85	79	80	78	77	82	103	100	96	99	93	93	93	90	95	100	99	99	100	99	99	100	99	99
49	P	SENTHIL KUMAR	48	M	78	LEFT COLLOID GOITRE	LEFT HEMITHYROIDECTOMY	2	70	62	77	83	80	81	89	88	86	125	120	110	121	105	100	94	90	88	71	68	60	69	61	58	57	60	61	89	85	77	86	76	72	69	70	70	100	98	100	100	98	99	100	99	98
50	E	SHAHEENA MARY	48	F	58	AC DISLOCATION	ORIF	2	92	88	86	94	96	93	98	95	91	131	128	132	130	128	125	122	127	129	80	78	75	83	81	82	77	79	74	97	95	94	99	97	96	92	95	92	100	99	100	100	99	100	100	98	99
51	P	ABHIRAMI	43	F	64	CA BREAST	LT MRM	2	79	70	84	88	90	86	83	91	89	138	130	120	124	116	114	109	100	99	90	86	90	80	74	72	70	71	68	106	101	100	95	88	86	83	81	78	98	99	99	98	99	100	98	99	99
52	E	UMA	44	F	59	CHRONIC SINUSITIS	FESS	2	78	66	78	80	83	79	81	80	79	130	127	125	131	126	120	121	119	111	70	71	76	70	66	68	69	72	75	90	90	92	90	86	85	86	88	87	98	100	99	98	99	98	99	100	
53	P	MANI	23	M	64	DEVIATED SEPTUM RT SIDED	SEPTOPLASTY	1	76	68	77	83	89	87	90	94	96	114	110	111	110	90	86	88	90	99	60	62	68	60	61	58	55	58	58	78	78	82	77	71	67	66	69	72	98	100	98	98	100	99	98	100	100
54	E	SIVASAMI	46	M	70	RIGHT COLLOID GOITRE	RIGHT HEMITHYROIDECTOMY	2	74	70	78	80	79	78	73	71	74	130	127	1123	129	120	119	121	124	117	80	76	74	81	77	79	73	75	79	97	93	424	97	91	92	89	91	92	99	99	98	99	99	98	99	100	99
55	P	DEEPA	34	F	60	MULTINODULAR GOITRE	SUBTOTAL THYROIDECTOMY	2	85	74	86	88	95	97	89	88	90	124	121	106	110	100	94	101	98	90	66	61	60	57	59	60	65	66	70	85	81	75	75	73	71	77	77	77	99	99	98	99	99	98	99	99	99
56	P	SURESH	29	M	58	FRACTURE NASAL BONE	REDUCTION	2	76	73	80	83	87	90	89	91	86	137	131	120	126	117	106	100	96	94	75	70	68	60	604	58	50	53	66	96	90	85	82	442	74	67	67	75	100	98	99	100	98	98	100	99	98
57	E	SHANTHA	46	F	63	IVDP L4-L5	DISCECTOMY	2	93	88	92	95	94	88	89	93	91	114	110	119	124	117	114	118	110	114	63	60	64	58	59	57	60	61	63	80	77	82	80	78	76	79	77	80	100	98	99	100	98	99	100	98	98
58	P	LOGANAYAKI	42	M	70	LT CA BREAST	MODIFIED RADICAL MASTECTOMY	1	77	70	79	81	93	97	91	90	88	129	120	117	126	111	100	105	97	90	74	70	68	61	71	60	55	59	60	92	87	84	83	84	73	72	72	70	99	98	100	99	98	99	99	98	98
59	P	GANDHIMATHI	50	F	63	PAROTID TUMOR	SUPERFICIAL PAROTIDECTOMY	2	89	84	93	99	101	99	103	98	91	127	125	111	109	104	99	94	86	88	71	68	60	66	60	55	59	60	63	90	87	77	80	75	70	71	69	71	98	99	100	98	99	100	98	98	99
60	E	MANIKANDAN	28	M	60	CHRONIC TONSILLITIS	TONSILECTOMY	1	80	74	71	78	81	79	84	77	83	121	1120	119	127	118	112	110	111	115	70	72	77	70	73	70	69	73	72	87	421	91	89	88	84	83	86	86	98	99	100	98	99	100	98	99	99